Notes On Clinical Examination

History Taking

Personal data: 1. Names, 2.Age, 3.Nationality 4.sex, 5.Adress 6.occupation, 7. Date of admission [Day with Time at am or pm] and the cause of the admission 8. Known illness and their duration.

Main complaints or problems:
List all of the patient's medical problems, preferably in the patient's own words.
List symptoms oldest first, and then the less old until the most recent symptom.

History of present complaint (HPC):
Include the answers to the direct questions concerning the system of the presenting complaint.
Timing, Location, Quality, Quantity, Aggravating and Relieving factors
How the disease is affecting patient's life (diet, sleep, clothing, bathing.....).
Describe past diagnostic testing, interventions and response to treatment if given.

Review of systems (ROS):

1- General: 1.fever, 2.night sweating

2- Cardiovascular system (CVS)

1. Dyspnea (breathlessness), 2. Orthopnea (breathlessness when lying flat), 3. Paroxysmal nocturnal dyspnea (attacks of nocturnal breathlessness), 4.Chest pain, 5.Palpitations, 6.Syncopal attacks (attacks of loss of consciousness) , 7.Ankle swelling, 8. Claudications (Lower Limb pain on exertion)

3- Respiratory system (RS)

1. Cough, 2.Sputum, 3.Haemoptysis (cough of blood), 4.Dyspnoea, 5.Wheezing, 6.Chest pain, 7.Exercise tolerance.

4- Gastrointestinal system (GIT)

1. Appetite, 2.Diet, 3.Weight, 4.Nausea, 5.Vomiting, 6.Haematemesis (vomiting of blood), 5.Dysphagia (difficulty with swallowing), 6. Odynophagia (pain with swallowing), 7. Heartburn, 8.Abdominal pain, 9.Abdominal distension, 10. Jaundice, 11.Bowel habit, 12.Nature of stooi, 13.Rectal bleeding, Mucus, 14.Incontinence, 15.Tenesmus.

5- Urogenital system (UGS)

1. Dysuria (painful micturition), 2. Suprapubic pain, 3.Loin pain, 4.Frequency of micturition including nocturnal frequency, 5.Urgency, 6.Haematuria (blood in urine), 6.incontinence.
In Male 1.Poor stream, 2.Dribbling, 3.Hesitancy (difficulty in starting to pass urine),
In woman 1.Date of menarche or menopause, 2.Frequency of menses, 3.Quantity and duration of menstruation, 4.last menstrual period,

6- Nervous system (NS)

1. Headaches, 2. Fits (Loss of consciousness), 3.Muscle weakness, 4.Sensory disturbances tingling and numbness (Paraesthesiae), 5.Smell, Vision, Hearing, Taste, 6.Tremor.

7- Musculoskeletal system (MSkS)

1. Aches or pains in muscles, bones or joints, 2.Swelling joints, 3.Limitation of joint movements.

8- Endocrine:

1. Polydepsia, 2.Polyphagia, 3.Weight gain or loss, 4.Menses duration, amount of flow, 5.Galactorrhea, 6.Weather tolerance, and 7.Proximal myopathy.

9- Hematology:

1. Easy bruising, 2.Gum bleeding, 3.Epistaxis, any bleeding from any body orifice, 4.Recurrent infections, 5.symptoms of anemia.

Previous Medical History (PMH): 1.History of similar illness, 2.Previous illnesses [DM, HT, Asthma, IHD, TB, Rheumatic fever, Epilepsy] 3.Previous Admissions, 4.Blood transfusion, 5.Operations or accidents, 6.Allergies for foods or drugs

Surgeries 1 2 3	Injuries 1 2 3	Hospitalizations 1 2 3	Blood Transfusion 1 2 3
Date		Indication	Date
Procedure		Facility	Units
Complications		Diagnosis	Complications
Improvement			

Drug history: ask about current and past medications and specifically about: 1. Steroids, 2. Aspirin, 3. contraceptive pill, 4.Anti-depressants.
For each drug ask about Prescribed by whom, Dose, Interval, Duration, side-effects, compliance.

Family history (FH): 1.Causes of death of close relatives, 2.Familial illnesses [DM, HT, Asthma, IHD, TB, Epilepsy]

Social history (SH): 1.smoking [Number of cigarettes per day] 2.Alcohol Drinking, 3.Drug abuse and tattoos, 4.Marital status, 5.Living accommodation, 6.Occupation, 7. Level of education, 8.Travel abroad in the past 6 months, 8.Sexula activities

History for all complains of pain: TLQSA

1- Timing: Onset, Duration, Diurnal variation, Frequency and Course
2- Location: Site, Radiation, Referral
3- Quality: Sharp stabbing or Dull aching, Compressing
4- Severity: Interference with Daily activity, Sleep, or Work
5- Aggravating and Alleviating factors, & Associated symptoms

History of all other complains: TQSA

1- Timing: Onset, Duration, Diurnal variation, and Course
2- Quantity: Frequency
3- Severity: Interference with Daily activity, Sleep, or Work
4- Aggravating and Relieving factors

History for any thing that gets out from the body from any orifice:

AS CBC

A= Amount
S= Smell
C= Color
B= Blood
C= Consistency

Note: don't use medical terms when telling history [don't say dyspnea but say breathlessness]
Note: don't say CBC say complete blood count

Skills essential for passing the exam

"How to look and act professional"

1- RELAX RELAX & RELAX [holding your breath and Quran will help] and SLEEP well the night before the exam

2- Dressing for the exam: [First impression is the last impression]
   a. For males: NO JEANS PLEASE and if you wear a suit, a **neck tie** and dark shoes it will be perfect and have your hair cut day before the exam.
   b. For female: Most important is the NAILS [no long nails], and please don't wear sport shoes.
   c. You should be wearing a hand watch and leave your mobile at home or in the car.

3- When you enter the room **Great** the examiners as well as the patient, and keep a **good eye contact** with both examiners through the exam and when you leave the room **Thank** the examiners and the patient "No matter how angry you are".

4- Fatal statements if you say them you will FAIL no matter how prepared and clever you and these are:
   a. هكي قالها الدكتور الفلاني
   b. انا قرتها في الكتاب الفلاني هكي
   c. انا هكي تعرفها

NEVER ARGUE WITH THE EXAMINER

لما المتحن يقولك حاجة و انت تعرف و متأكد ان هي غلط رد بالك تحساب ان الممتحن ميعرفش المعلومة الصح و لكن هو في هذه الحالة يشوف في حتر مك و تصديقك للي اكبر منك (مش علمك).
لما يقولك الممتحن اي حاجة ما عمرك ما تناقش معاه و الانطباع اللي توريه هو انك معجب بالعلم اللي يقولك فيه و قداش انت ما تعرف شي و هو العلم اللي يقول فيه حاجة صقع. كل ما تنجح في توصيل هذا الانطباع كل ما تزيد فرصتك في النجاح.

5- If you don't know the answer say "I don't know" [never make up answer] because that question mark is already lost so why to lose time in addition to the lost mark.
رد بالك تكذب مهما حسيت ان الحاجة اللي مادرتهاش مهمة لأن الممتحن غالبا يعرف الحالة و طبعا كان عرفك تكذب انسي انك تنجح

6- The examiner will always ask you something that you don't know and your response should be "I don't know". The examiner will write down that you admit the lack of knowledge = not an arrogant doctor and you gain a mark.

7- Always be polite and thank the examiner no matter how aggressive he was with you.

8- You should bring your stethoscope, thermometer, sphygmomanometer, hand watch, torch, tongue depressor, tape measure and regular ruler, tendon hammer, cotton, sterile needle, & if you have tuning fork bring it.

9- If you can speak in English only, please do.

10- If you say a wrong answer and you realize that while saying it, retract your statement.

11- There is **only one way** to master examination "examine pt repeatedly in front of an observer" the observer may be your friend.

General Examination Checklist

Action	YES	NO
Permission taken		
Stood on the Right side of bed		
Vital signs [4]		
Observation		
General appearance		
Position of the patient on bed		
Mental state		
Emotional state		
Build		
Comment on pt surrounding		
Face		
Symmetry		
Skin discoloration or disease		
Eyes for jaundice		
Asked pt to look to the window		
Eyes for pallor		
Pupil size of both eyes		
Mouth		
Cyanosis		
Pallor		
Ask pt to turn his tongue up		
Jaundice		
Odor		
Use torch and spatula		
Ulceration		
Buccal pigmentation		
Tongue appearance		
Teeth and Gum		
Soft palate and tonsils		

Action	YES	NO
Neck		
Sit up the patient		
Symmetry, swellings, Scar		
Lymphatic glands		
Thyroid gland		
Trachea		
JVP		
Upper limbs		
Nails: Clubbing & splinter hge		
Nails: Pallor & Koilonychia		
Palms Muscle wasting		
Palmar creases		
Palmar Erythema		
Pulse [6 elements]		
Axiliary lymph nodes		
Lower limbs		
Swelling, Scar, Varicosity,		
PVD changes [ulcer, hair loss]		
Look for interdigital infection		
Peripheral pulses		
Temperature		
Calf muscle tenderness		
Test for pitting Edema		
Ethics		
Cover back the patient's legs		
Thank the patient		

General Examination

Permission is very very important

Stand on the right side of the patient **ALWAYS**

Vital signs

1- Heart rate in beats/min. tachycardia if > 100 b/min and bradycardia if < 60 b/min [Count the rate for one minute and write down an odd number (عدد فردي) by increasing or decreasing one number so the examiner will know that you counted for 1 min]
2- Respiratory rate in breath/min. Tachypnea if >20 breaths/min
3- Temperature. You must note were you measured it e.g. [37.4 measured at the right axilla] Fever is temperature > 37.5
   For each 1 degree ↑ in Temp→ ↑ 13 beats/min in HR and 4 breaths/min in RR
4- Blood Pressure: measure it in both arms and comment on how you measured the diastolic pressure

The patient HR was 77 beats/min, RR of 16 breaths/min, Temperature was 37.7 measured at the right axilla, and BP was in both arm 120mmHg systolic over 80mmHg diastolic measured on disappearance of the sound.

General

1- General appearance (does the patient look healthy= well, unwell, or ill)
2- Mental state (oriented or confused).
3- Emotional state (cooperative or not). In the exam don't say any bad thing about the patient so always try to say that the patient is cooperative
4- Build (underweight, average, overweight), and comment whether the increase in weight is due to edema or obesity.
5- Skin color: cyanosis, jaundices, pallor, plethoric, pigmentary change, and comment on vitilligo, petechie or spider navei if present.
6- Features of endocrine disease [Cushinoid or Acromegalic].
7- Comment on any nebulizer, cannula, catheter, infusion pump, or I.V fluid, and the chart, wheel chair or walking stick.

The comment: Mr. (tell the pt name)..... is ...position... on the bed, he looks ....General appearance..., and he is ...mental status...and ... cooperation..., he is ...built...He is ...color...he has ....cannula or other .....

E.g. Mr. Ali is lying on bed comfortably he looks unwell, he is oriented and cooperative, Mr. Ali is overweight, he has cannula inserted in the dorsum of his left hand, and it is connected to an infusion pump and there is a face mask connected to oxygen slender beside the patient

Face

1- Inspection: symmetry [Asymmetrical in facial nerve palsy], rashes [Malar rash in Mitral stenosis and SLE], Acne, features If endocrine disorder.
2- Eyes:
   - Yellowish discoloration of the sclera and pallor of the conjunctivae.

   When you look for jaundice in the sclera **ask the patient to look toward the window for natural sunlight.**

   Jaundice definition: It is the yellowish discoloration of skin, sclera and mucous membrane due to the accumulation of bile pigments.

   Why Bilirubin precipitates in Sclera?

   Because binds to elasin protein and there is high concentration of this protein in the sclera
   - The eyes may be red due to conjunctivitis and in this case they are not reliable as a site for looking for anemia
   - If there is Ptosis comment on it

Mr/Mrs...face is symmetric and there is no rash, the conjunctivae are not yellow and not pale, the pupils looks equal

3- Mouth and Pharynx:

   Torch and tongue depressor should be used
   - Breath odor
   - Ulcers
   - Lips: color and eruption
   - Tongue: appearance in terms of color [bluish in central cyanosis] and smoothness [iron deficiency anemia and Vit B12 deficiency anemia].

     Ask the pt turn his tongue so you can look at the undersurface of the tongue for jaundice
   - Buccal mucosa: color and pigmentation [DDx Addison's disease and Peutz-Jegher syndrome] look for pigmentation specifically on the hard palate.
   - Teeth and gums: hygiene, if using denture comment on it
   - Movement of the soft palate and the condition of the tonsils, by asking the pt to say ahhhh.

Breath odor DDx	Ulcers DDx
1- Halitosis [poor hygiene, bronchiectasis and lung abscess] 2- Acetone→DM 3- Ammoniac→Uremia 4- Alcohol 5- Hepatic fetor "fishy' 6- Feculent→ intestinal obstruction	1- Aphthous ulcer "Crohn's disease and SLE"" 2- Herpes simplex 3- Pemphigus 4- Tumor

Neck

1- Inspection: Symmetry, swellings, Scar [LN biopsy or Thyroidectomy surgery] and dilated veins.
2- Palpation: Lymphatic glands , Thyroid gland and Trachea.
3- Measurement: JVP.

Q-If you feel LN what should you do next?
You should palpate for Axillary and Inguinal lymph nodes, and palpate for hepatosplenomegaly.
The LN is a mass and should be described as any mass: Number, Site, Size,

> E.g. The neck is symmetric there is no swelling, no visible veins, no scars, no lymph node is detected by palpation, thyroid gland is normal, JVP is ... cm, trachea is central.

For JVP see cardiovascular examination. For thyroid see thyroid examination

Upper limbs

1- Nails: Clubbing, Pallor, Koilonychia (flat or concave), leukonychia(indicates hypoalbuminemia, Splinter hemorrhage (trauma, rheumatoid arthritis, infective endocarditis) , Psoriasis (onycholysis, thickening of the nails, pitting, yellow oil drops).
1- Palms for Erythema [RA, ↑T4, Polycythemia, Pregnancy] or muscle wasting.
2- Palmar creases for anemia
3- Pulse: 1.rate 2.rhythm 3.character 4.volume 5.synchronous (radioradial and radiofemoral) and 6.the condition of the wall
4- Axillary lymph nodes

Lower limbs

1- Inspection: swelling, scar, varicose veins, interdigital infection & changes of peripheral vascular disease [ulcer, atrophic shiny skin, loss of hair].
2- Palpation:
    - Is the Edema of feet or ankles pitting or not.
    - Peripheral pulses.
    - Temperature.
    - Calf muscle pain.

Generalized Edema

Definitions:

Edema: A clinically apparent increase in the interstitial fluid volume.
Anasarca: gross, generalized edema.
Ascites: accumulation of excess fluid in the peritoneal cavity.
Hydrothorax: accumulation of excess fluid in the pleural cavity.

Causes of generalized edema:

1- Cardiac failure
2- Liver Cirrhosis
3- Nephrotic syndrome
4- Malnutrition
5- Hypothyroidism
6- Pregnancy
7- Drugs → Calcium channel blockers, NSAID, andCCP

Causes of unilateral LL edema:

1- DVT
2- Cellulitis
3- Ruptured Baker's cyst

Q- What are the types of edema?
They are 2 types:

1- Pitting edema: reflects increased interstitial fluid, .occurs in all causes except lymphedema, and hypothyroidism (myxedema)
2- Non-pitting edema: reflect protein deposition lymphedema and hypothyroidism (myxedema)

Q- What are the sites to look for edema?

1- Face for Periorbital edema
2- Eyes for Papilledema by ophthalmoscope
3- Fingers
4- Abdomen for Ascites
5- Over the sacrum in a bed ridden patient
6- Scrotum
7- Lower limbs

Cause	Cardiac Failure	Liver cirrhosis	Nephrotic syndrome
Pathophysiology	1- ↑ Hydrostatic pressure 2- Hypoalbuminemia after causing cardiac cirrhosis 3- Hyperaldosteronism due to activation o f RAAS	1- Portal hypertension→ ↑hydrostatic pressure 2- Hypoalbuminemia 3- Hyperaldosteronism due to ↓ destruction→Renal Na retention 4- ↑estrogen due to ↓ destruction → Renal Na retention 5- Lymphatic obstruction	1- Hypoalbuminemia
History	Dyspnea with exertion prominent—often associated with orthopnea—or paroxysmal nocturnal dyspnea	Dyspnea infrequent	May be associated with uremic signs and symptoms
Examination	• ↑JVP, S3 and S4, and hepatomegaly • Bilateral lower limb edema > Ascites • Pulmonary edema	• JVP N or ↓ • Ascites > edema of lower limbs and scrotum • Signs of chronic liver disease	• Periorbital edema noted on awakening • ↑ BP
Management	↓ Na intake and diuretics	↓ Na intake and diuretics	↓ Na intake and diuretics

Causes of pleural effusion = Causes of Ascites:

1- Cardiac failure
2- Liver cirrhosis
3- Nephrotic syndrome
4- Infections→ TB or other bacteria
5- Malignancy

Generalized Lymphadenopathy

Definition:
It's a lymphadenopathy that involves 2 or more non-contiguous sites

Submandibular nodes <1 cm and inguinal nodes of < 2 cm, are considered normal

DDx:

1- Viral→ infectious mononucleosis syndromes (EBV, CMV) and HIV
2- Bacterial→ TB, Syphilis
3- Parasitic→Toxoplasmosis
4- Immunologic diseases→ RA, SLE and Sarcoidosis
5- Malignant diseases→ Hodgkin's disease, non-Hodgkin's lymphomas, and leukemias or secondaries for a solid tumor
6- Drug→ Phenytoin

Groups of lymph nodes: "examine them in the following order"

1- Cervical: Submental→ Submandibular→ Anterior cervical→ Posterior cervical → Preauricular→ Posterior auricular→Occipital → Supraclavicular
2- Axillary: Anterior gp→ Posterior gp→ Medial gp→ Lat gp→ Apical
3- Epitrochlear LN: enlarged mainly in EBV and in Syphilis
4- Inguinal

Examination of Cervical Lymph Nodes Swelling = Lateral neck swelling

Scenario: Examine this pt neck

- Inspection: Site, if there is overlying Erythema, Scar" biopsy" or Discharge
- Palpation:
    - Temperature and Tenderness → Number "matted or not → Size→consistency→ attachment to skin or underlying structures
    - Palpate for thyroid gland and centrality of the trachea
    - Say I would like to measure the JVP

E.g. By inspection there is a swelling in the Rt anterior triangle of neck, there is no redness of the overlying skin, no scar and no discharge
By Palpation it's not hot and not tender, there is/are...number... about ...by...cm in size they are firm in consistency and not attached to skin or overlying structure

Q- What do like to do next?
I like to look at the pharynx →Why? → Because the most common cause of cervical lymphadenopathy if URTI and dental carries—Ok what else you would like to examine→ other groups of lymph nodes and for hepatosplenomegaly because all are a part of Reticuloendothelial system

Q- What is the name of left supraclavicular lymph node and what is its importance?
It's named Virchow's and it's enlarged in malignancy of the GIT mainly the Stomach cancer.

Q- If the patient is having night sweating what might be the cause?
Hodgkin lymphoma or TB

Q- What are Ix you like to do?
Laboratory tests:
1- CBC, ESR and CRP Neutrophila in bacterial infection and Lymphocytosis in viral infection
2- Throat culture
3- Monospot test = Paul-Bunnel test for EBV infection
4- Blood film→ EBV and Leukemia

Radiological Ix
1- Chest x-ray for mediastinal lymphadenopathy → TB or Sarcoidosis
2- Abdominal ultrasound to look for paraaortic lymphnodes

Histopathological Ix
1- Excisional lymph node biopsy → to exclude malignancy

CVS important questions in History

Chest pain

As regular TLQSA

Ask about other symptoms of respiratory system and cardiovascular system

Additional questions in chest pain

1. Questions for anginal pain → "Does the pain occur at rest? Exertion? Exposure to cold? Emotional strain?"
2. Chest pain ↑ with inspiration: 5Ps 1-pneumothorax 2-pleurisy 3-pneumonia 4-pericarditis 5- PE
3. Pain relation to position→ pericarditis better when you lean foreword because you relax the pericardium [also pancreatitis pain is relieved by leaning foreword but its pain is epigastric]
4. Ask about GERD → heart burn and the relationship to eating?

Dyspnea – Abnormal awareness of breathing

As all complains: TQSA

Ask about other symptoms of respiratory system and cardiovascular system

Additional questions in dyspnea:

1- Does the shortness of breath occur with exertion? at rest? lying flat? sitting up?

2- How many flights of stairs can you pt raise without becoming short of breath? And How many level blocks could you walk 6 months ago?

3- Classify the breathlessness by NYHA classification

NEW YORK HEART ASSOCIATION (NYHA) CLASSIFICATION	
NYHA class	Breathlessness
I	No symptom on ordinary exercise
II	On Ordinary exercise, "Stairs"
III	On less than ordinary exercise "patients develop shortness of breath on activities of daily living such as having a shower, etc. "going to bathroom"
IV	At rest

Orthopnea – breathlessness ↑ when pt lies down

Inquire of all patients, "How many pillows do you need to sleep?" and comment as the following."3-pillow orthopnea for the past 4 months".

PND – sudden breathlessness after period of sleep

Palpitation

As all complains: TSA

Ask about other symptoms of cardiovascular system

Ask about symptoms of Thyrotoxicosis

Ask the pt to tap the rhythm

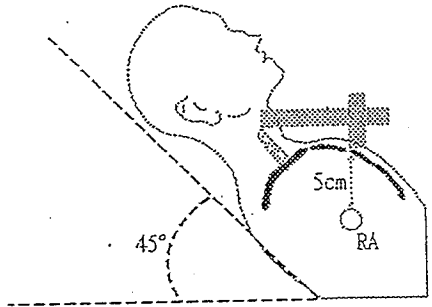
Ask if the patient had to urinate after the attack

Syncope → the of your history will be that is it a real syncope or it was seizure

Syncope	Seizure
Occurs after arising from lying or sitting position	Seizures typically not related to posture.
Pallor	cyanosis
The duration of unconsciousness is usually very brief (i.e.,seconds)	The duration of unconsciousness is prolonged (i.e., > 5 min) in a seizure
Do not occur	Incontinence
Do not occur	Post-ictal confusion and headache

Examination of the JVP

The patient should be in **45°** & the **height** and **waves** of the JVP should be assessed. The head should be supported on a pillow and turned slightly to left to see the right internal jugular vein. Observe medial to the sternomastoid muscle.



Q. What are the difference between JVP and Carotid pulse by examination?

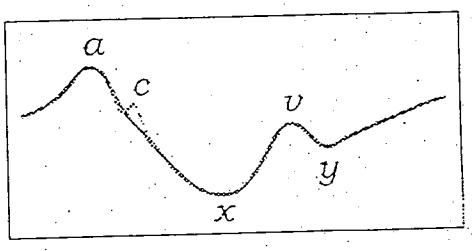
JVP	Carotid pulse
More visible than palpable	More palpable than visible
Obstructed by finger pressure	Not obstructed
Varies with posture	No
When venous pressure is high it moves ear lobe	Does not move ear lobe
Decrease with respiration	No
Tow waves of cardiac cycle	Only one per cardiac cycle
More prominent when put pressure on abdomen [hepatojugular reflex]	No effect by Abdominal pressure

Assessment of the height

It is normally less than 3 cm. This equates to a right atrial pressure of 8 cm of water as in this position, the manubriosternal angle is about 5 cm above the centre of the right atrium.

Raised JVP (> 3 cm) causes are:

1- Right-sided heart failure.
2- Tricuspid or Pulmonic valve disease
3- Constrictive pericarditis & Pericardial tamponade.
4- Obstruction of superior vena cava.
5- Fluid overload e.g. renal disease.



Waveforms of the JVP

Waves

- *a* - presystolic; produced by right atrial contraction
- *v* - occurs in late systole; increased blood in right atrium from venous return

Descents

- *x* - atrial relaxation
- *y* - tricuspid valve opens and blood flows in to the right ventricle

Abnormalities of the JVP

- Absent a wave → atrial fibrillation
- Dominant a wave
  1. Pulmonary stenosis 2. Pulmonary hypertension 3. Tricuspid stenosis

- Cannon a wave
  1. Complete heart block
  2. Ventricular tachycardia
- Dominant v wave →Tricuspid regurgitation

JVP normally falls with inspiration but may rise (**Kussmaul's sign**) in constrictive pericarditis. [NOT pericardial tamponade]

Examination of the pulse

Rate: normal is from 60-100 beat/min. if<60 its bradycardia, and if >100 its tachycardia
Rhythm:

- Regular→ Normal
- Regularly irregular→ sign of bigeminy or trigeminy (occurs in ectopic ventricular beats, or digitalis toxicity)
- Irregularly irregular→ **Atrial fibrillation**

Volume: small, good, or large

- *Small volume pulse (pulsus parvus):* AS, MS, HF, IHD, & pericardial tamponade
- *Large volume pulse:* same as those of collapsing pulse

Character:

- *Slow rising pulse* (*pulsus tardus*): Delayed upstroke (aortic stenosis).
- *Bounding = collapsing (hyperkinetic) pulse*: aortic regurgitation and Hyperkinetic circulation [fever, anemia, thyrotoxicosis, pregnancy].
- *Pulsus bisferiens*: Double systolic pulsation in mixed AR & AS.
- *Pulsus alternans*: Regular alteration in pulse pressure amplitude (severe LV dysfunction).
- *Pulsus paradoxus*: Exaggerated inspiratory fall (>10 mmHg) in systolic bp (Pericardial tamponade, severe asthma).
- *'Jerky' pulse*: hypertrophic obstructive cardiomyopathy

Precordium Examination Checklist

Action	YES	NO
Permission taken		
Stood on the Right side of bed		
Put pt in 45° [semi-recumbent]		
Adequate Exposure		
Inspection		
Discoloration		
Deformity		
Superficial veins		
Scar		
Apical impulse		
Visible pulsation in all valves		
Visible heave		
Visible pulsation on epigastrium		
Palpation		
Apical beat position		
Thrill in all valve areas		
Heave in Lt parasternal area		

Action	YES	NO
Auscultation		
Warm the stethoscope		
Timing by palpating Rt carotid		
With diaphragm on 4 area		
Bell on mitral & tricuspid area		
Bell on mitral in Lt Lat position		
Diaphragm on Lt axilla		
Diaphragm on Rt carotid		
Listen for change with inspiration		
Listen for changes with expiration		
Ask the patient to Sit		
Diaphragm on Lt 3rd ICS at expiration		
Auscultate lung bases		
Palpate the liver edge		
I would like to do the following		
Check for BP, JVP, Peripheral pulses, & lower limb edema		
Ethics		
Dress the patient back		
Thank the patient		

Precordium Examination

Inspection

Forceful apical pulsation → Left ventricular hypertrophy
Left parasternal pulsation → Right ventricular hypertrophy
Lt 2nd ICS pulsation → enlarged pulmonary artery
Rt 2nd ICS pulsation → aortic aneurysm
Diffuse apical pulsation → cardiomyopathy or post MI
Double beat pulsation → HOC
Epigastric pulsation → 1- normal in thin person, 2- Rt ventricular enlargement, 3- Abdominal aneurysm, 4- Pancreatic cyst, 5- Pulsatile liver in TR

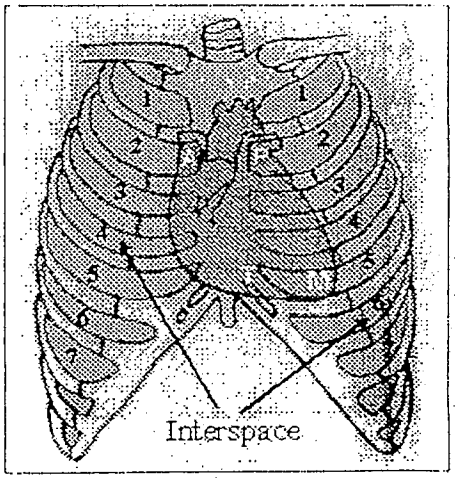
Palpation

- **Apical beat definition**: most downward & most lateral palpable beat
- Impalpable beat occurs in:
    1. Obesity
    2. Heart under the rib
    3. Pericardial effusion
    4. Deviation of the mediastinum due to lung or pleural disease
    5. Dextrocardia

- Apical beat possibilities:
    1. Normally as gentle non-sustained
    2. Displaced due to left ventricular enlargement
    3. Forceful = Sustained = thrusting indicates a pressure overloaded left ventricle such as in hypertension or aortic stenosis.
    4. Tapping in MS
- Palpation for Lt parasternal heave which indicates Rt ventricular hypertrophy
- **Heave or lift** = more vigorous apical impulse than expected

Location of the valve area

Aortic: 2nd R ICS at right sternal border
Pulmonic: 2nd L ICS at left sternal border
Second Aortic: 3rd L ICS at left sternal border
Tricuspid: 4th L ICS along lower left sternal border
Mitral (apical): 5th L ICS at MCL



Auscultation

S1 + S2+ added sound + murmur

Uses of the bell: to listen for Mid-diastolic murmur of MS, S3 & S4

S1

- Heard with diaphragm, loudest at apex or at lower left sternal border
- Due to closure of the mitral valve

•*Loud* S1: Mitral stenosis, hyperkinetic heart (anemia, fever, hyperthyroidism), thin chest wall.
•*Soft* S1: Mitral regurgitation, heart failure, thick chest wall, pulmonary emphysema.

S2

- *S2* heard at Lt 2nd ICS with the diaphragm
- Its due to closure of Aortic & Pulmonic valves so it has 2 components

• *Loud* A2: Systemic hypertension.
• *Soft* A2: Aortic stenosis (AS).
• *Loud* P2: Pulmonary arterial hypertension.
• *Soft* P2: Pulmonic stenosis (PS).

Added sounds

S3

- Low-pitched, heard best with bell of stethoscope at apex, following S2
- Caused by rapid ventricular filling
- normal in children, pregnant and adults < 35 years;
- After age 35, S3 indicates LV failure or volume overload as in MR or AR.

S4

- Low-pitched, heard best with bell at apex, preceding S1.
- Caused by atrial contraction into a noncompliant ventricle.
- Found in AS, hypertension, HCM, IHD, & acute MR.
- S4 is Absent in atrial fibrillation

Opening Snap (OS) High-pitched; follows S2, heard at lower left sternal border and apex in mitral stenosis (MS); the more severe the MS, the shorter the S2–OS interval.

Ejection Clicks High-pitched sounds following S1; occurs in, congenital AS (loudest at apex) or PS (upper left sternal border).

Midsystolic Clicks At lower left sternal border and apex, often followed by late systolic murmur in mitral valve prolapse.

Heart Murmurs

Murmurs are **not** added sound

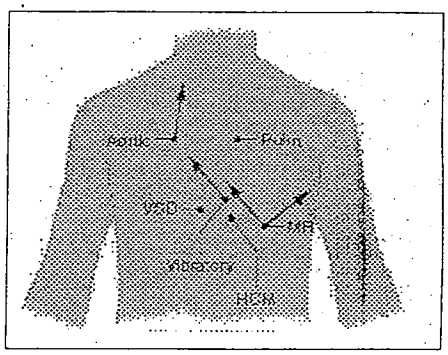
SYSTOLIC MURMURS Ejection-type : • Aortic valve stenosis • Hypertrophic obstructive cardiomyopathy • Aortic flow murmur • Pulmonic valve stenosis • Pulmonic flow murmur Pansystolic: • Mitral regurgitation • Tricuspid regurgitation • Ventricular septal defect Late-systolic: • Mitral or tricuspid valve prolapse
DIASTOLIC MURMURS • Early diastolic Aortic valve regurgitation • Pulmonic valve regurgitation • Mid-to-late diastolic Mitral or tricuspid stenosis
CONTIOUS MURMURS • Patent ductus arteriosus

Comment on the 4 characteristics of murmurs:

1. Timing
2. Grade = Intensity
3. Location and radiation (axilla for MR carotid for AS, liver for TR)
4. Variation with respiratory phase, standing, or Valsalva maneuver

Intensity of murmur

Grade I: just audible in a quite room, with patient's breath held, never immediately
Grade II: Quite
Grade III: Moderately loud
Grade IV: Loud and accompanied by thrill
Grade V: very loud with only the rim of the diaphragm is need to hear the murmur
Grade VI: heard with stethoscope over the chest



Location & Radiation

Location (see above)
Radiation: some systolic murmurs has radiation
MR→ radiates to the left axilla or the back
AS→ radiates to carotids

	MS	MR	TR	AR	AS
Pulse	Small volume pulse	-	-	*collapsing pulse or Pulsus bisferiens*	Slow rising pulse *[Pulsus Tardus]*
Inspection	Malar rash	Visible pulsation at the apical area	Visible pulsation over the epigastrium	Visible pulsation over apical area, Aortic area ± over the root of the neck	Visible pulsation Over the apical areas
Palpation Apical beat:	Not displaced Tapping apical beat (palpable S1) ± Lt parasternal heave	**Displaced** downward & laterally ± Lt parasternal heave	Normal Lt parasternal heave	**Displaced** downward & laterally Forceful	Not displace Forceful = Thrusting
Thrill	murmur is ≥ G4	murmur is ≥ G4	murmur is ≥ G4	murmur is ≥ G4	murmur is ≥ G4
Auscultation					
S1	Loud	Soft	Soft	N	N
S2	N or ↑	N or ↑	N	N	Soft
Added sound	Opening snap	S3 over mitral area	S3 over tricuspid area	Ejection click ± S3+S4	Ejection click
	Rumbling mid-diastolic murmur over the mitral area it about G3-4 there is no radiation, and ↑ with expiration and ↓ by inspiration	**Pansystolic** murmur over the mitral area its Grade is about 3-4 its radiating to the axilla, there is no hepatic pulsation, its ↑ with expiration and ↓with inspiration	**Pansystolic** murmur over the tricuspid area its Grade is about 3-4 its not radiating to the axilla, there is hepatic pulsation, its ↓ with expiration and ↑ with inspiration	There is **early diastolic** murmur over the aortic area its grade is about 3-4, no bruit can be heard over the carotids. Significant AR can be accompanied by systolic flow murmur	**systolic ejection murmur** over the aortic area its grade is about 3-4, its radiating to the carotid arteries its ↑ with expiration and ↓with inspiration

Complications for all murmurs:

1- Arrhythmias mainly AF
2- Embolisation
3- Of the left side → LVF → RVF
4- Of left side → pulmonary HT
5- Of Right side→ RVF→ hepatic cirrhosis

Investigation for all:

1. ECG
2. CXR
3. Echo

Treatment for all:

Explain to patient about his disease
Rx for arrhythmias and HF
Surgery if patient becomes symptomatic
If stenosis→ commisurotomy or balloon valvoplasty or replacement
If reguirge→ replacement

Valvular heart diseases

Stenotic Etiology:	Regurgitant Etiology:
1. Rheumatic fever. [Most common cause for MS]	1. Dilatation of ventricle
2. Congenital abnormalities.	2. Rheumatic fever.
3. Senile degeneration. [Most common cause for AS]	3. Infective endocarditis
	4. Post-MI with rupture of papillary muscles

Mitral Stenosis

The normal mitral valve area (MVA) is 4.0-6.0 cm^2 & pt become symptomatic when < 2 cm^2
Mild stenosis→ 1.5-2.5 cm^2
Moderate stenosis→1.0-1.5 cm^2
Severe stenosis → <1.0 cm^2

Etiology

1. **Rheumatic fever** MOST COMMON CAUSE
2. Congenital abnormalities (rare)

Clinical picture

Epidemiology: female > males, usually present at age 40-50 years
History: **only half** of the patients will give a history of rheumatic fever during childhood, typically the patient is **female** [female > males] which reaches 40 years old or becomes a **pregnant** start to develop the symptoms & signs of MS.

Symptoms & Pathophysiology

Progressive fibrosis & calcification of the valve leaflets→ MS → ↑Lt At pressure →

1. ↑ Pulmonary venous pressure → **Dyspnea** (most common presenting symptoms)
   → Rupture of capillaries →**Hemoptysis**
   → Pulmonary hypertension which may cause **chest pain**
2. Pulmonary hypertension→ RVH → **RVF** with all of its symptoms [review HF sheet]
3. Lt atrial enlargement →Pressure on the esophagus leading to (**Dysphagia**)
   →Pressure on Left recurrent laryngeal N→ hoarseness (**Ortner's syndrome**)
   →Arrhythmias (mainly AF)→ **Palpitations**
4. Abnormal hemodynamics → thrombus formation which when detach → **Thromboembolic phenomen** [CVA, Limb ischemia, Mesenteric infarction...]

Any factor that ↑ cardiac output it will → ↑ in Lt atrial pressure→ will make the symptoms appear or exacerbates the Already present symptoms

Signs

General examination:
Face → **Malar rash**
Neck →if RVF → ↑JVP
Hands → irregularly irregular pulse if AF is present
LL→ if RVF → LLE

Precordial examination: (see Table)
I→ if PHT → pulsation in pulmonary area, & If RVH →Lt parasternal heave.
P→ **Tapping apex beat**, Thrill if murmur ≥ 4 , & if RVH Lt parasternal heave.
A→ Loud S1 & normal S2 + low pitched **rumbling Mid-diastolic murmur** at mitral area heard best with the bell in left lateral position with patient in expiration with OS [**opening snap**]

Opening Snap High-pitched; follows S2, heard at apex in mitral stenosis (MS)
The more severe the MS, the shorter the S2–OS interval & when S1 & OS are inaudible it indicates that the valve is heavily calcified.
So the presence of opening snap indicates a mobile relatively good valve.

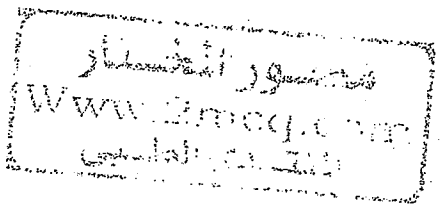
Investigations

ECG: may show AF, Left atrial enlargement, or RVH. [see ECG sheet]
CXR: may show Left atrial enlargement [Cardiomegaly, & splayed tracheal bifurcation], Kerley's lines
Echocardiography

- **Initial investigation of choice** because it Confirms the diagnosis & Assesses the severity of the stenosis

Management

- Pts should receive prophylaxis for rheumatic fever and infective endocarditis
- Asymptomatic patient just follow up with echocardiography every 6-12 months.
- Asymptomatic patient with mitral orifice < 1.7 cm2surgery percutaneous balloon valvuloplasty is indicated
- If the pt is symptomatic then percutaneous balloon valvuloplasty is indicated
- Diuretics to control pulmonary congestion & Warfarin to control thromboembolic complication
- In the presence of AF [see AF treatment sheet]
- Percutaneous balloon valvuloplasty is the procedure of choice; if not feasible, and then opens surgical valvotomy.



Mitral regurgitation

Etiology

1- Dilatation of ventricle
2- Rheumatic fever.
3- Infective endocarditis
4- Post-MI with rupture of papillary muscles

Clinical picture

- Symptoms & sign of LVF
- Symptoms & signs of RVF
- Precordial examination (see table)

Investigations

ECG: Left atrial enlargement, AF, RVH, or LVH.

CXR: may show Cardiomegaly due to Left atrium or Left ventricle

Echocardiography

- **Initial investigation of choice** because it Confirms the diagnosis & Assesses the severity of the regurgitation

Management

- Pts should receive prophylaxis for rheumatic fever and infective endocarditis
- Asymptomatic patient just follow up with echocardiography every 6-12 months.
- Afterload reduction (ACE inhibitors, hydralazine, or IV nitroprusside) decreases the degree of regurgitation, increases forward cardiac output, and improves symptomatology.
- Asymptomatic patient with LV dysfunction (LVEF _ 60% or end-systolic LV diameter by echo <45 mm) valve repair or replacement is indicated. & Operation should be carried out *before* development of severe chronic heart failure.
- If the pt is symptomatic then valve repair or replacement, is indicated.
- In the presence of AF [see AF treatment sheet]

Tricuspid regurgitation

Causes

1- **Infective endocarditis in drug abusers**
2- Right ventricular failure which leads to stretching of valve rings
3- Rheumatic heart disease

Clinical picture

It give the clinical picture of Right sided heart failure [see heart failure sheet]

Examination (see table)

- Jugular venous pulsation: A large v wave is seen in jugular veins during systole.
- Pulsatile liver: Systolic expansion of the liver frequently is present.

ECG:

- May show Right ventricular hypertrophy

Chest X-ray → May show Cardiomegaly due to right ventricular dilatation

Echocardiography: **Initial investigation of choice** because it Confirms the diagnosis & Assesses the severity of the regurgitation

Aortic Stenosis

The **Most Common** valve disease [at age of 80 years 10% of population has AS]
Anatomy: The normal aortic valve area is 3.5 cm^2.

- Mild stenosis→ 1.5-2.0 cm^2
- Moderate stenosis→ 1.0 1.5 -cm^2
- Severe stenosis→ <1 cm^2 (or a mean gradient of>50mmHg)

Etiology

1- **Congenital** aortic stenosis: it's very rare and it may be the result of bicuspid valve, or a subaortic membrane, constricting the left ventricular outflow tract.
2- **Premature calcification** of a **congenitally bicuspid aortic valve**: patients typically develop symptoms from age 40 years onward.
3- **Rheumatic fever**: always associated with mitral valve disease & usually develop in 4th to 5th decade
4- **Calcific aortic stenosis of normal valve**: it the most comm. Cause and occurs from age of 65 years onward.

Symptoms (*Classical Triad*)

1- **Effort dyspnea** [due to left ventricular failure]
2- **Effort angina** [severe LVH→ mismatch between O_2 supply & O_2demand] ± CAD
3- **Effort dizziness or syncope** [due to peripheral vasodilation → hypotension in the presence of a fixed cardiac output.]

- Pts may present with symptoms & signs of LVF

Examination →See table

Investigation
ECG:

- Left ventricular hypertrophy (usually)
- LBBB (due to calcification of the conducting system)

Chest X-ray → May show LVH or Calcifications of the aortic valve
Echocardiography: **Initial investigation of choice** because it Confirms the diagnosis & Assesses the severity of the stenosis

Management

- Avoid strenuous activity in severe AS, even in asymptomatic phase.
- Asymptomatic patient just follow up with echocardiography every 6-12 months.
- Asymptomatic patient with valvular gradient > 50 mmHg and with features such as left ventricular systolic dysfunction then consider surgery [Operation should be carried out *before* frank failure has developed].
- If the pt is **symptomatic** then **Valve replacement** is indicated [death usually occurs within 3-5 years of the onset of symptoms].
- Balloon valvuloplasty is limited to patients with critical aortic stenosis who are not fit for valve replacement
- Treat HF in standard fashion but *avoid afterload reduction [No vasodilators].*
- Statin therapy may slow progression of leaflet calcification.
- Advice for Endocarditis prophylaxis

Aortic Regurgitation

Causes

	Acute	Chronic
Dilatation of valve ring	Aortic dissection	Idiopathic **Ankylosing spondylitis** Hypertension Syphilitic aortitis Marfan's syndrome
Diseased valve cusps	**Infective endocarditis**	Congenital → bicuspid valve Acquired→ RF, SLE

More common in males

Symptoms

- Those of LVF [dyspnea, orthopnea, PND, fatigue]
- Angina may occur due to low diastolic pressure reducing coronary perfusion

Examination (Precordial examination→ (see table)

- *Bounding = collapsing (hyperkinetic) pulse*
- *Pulsus bisferiens*: Double systolic pulsation in mixed AR & AS.
- When aortic regurgitation is severe it may lead to the following signs:

Investigation

ECG:

- Left ventricular hypertrophy (usually)

Chest X-ray → May show Cardiomegaly due to LV dilatation or Aortic aneurysm

Echocardiography:

- Initial investigation of choice because it Confirms the diagnosis & Assesses the severity of the regurgitation

Management

- Asymptomatic patient just follow up with echocardiography every 6-12 months.
- Asymptomatic pts with LV dysfunction (LV ejection fraction<55% or LV end-systolic diameter < 55mm) **valve replacement** is indicated
- If the pt is **symptomatic** then **valve replacement** is indicated should be carried out in pts with severe AR when symptoms develop or in asymptomatic.
- Vasodilators (long-acting nifedipine or ACE inhibitors) may delay need for operation.
- Treat HF in standard fashion
- Advice for Endocarditis prophylaxis
- If the cause is RF give prophylaxis

Respiratory Examination Checklist

Action	YES	NO
Permission taken		
Stood on the Right side of bed		
Adequate Exposure		
Inspection		
Deformity		
Discoloration		
Scar		
Swelling		
Superficial veins		
Inspection of chest movement		
Symmetry expansion		
Use of accessory muscles		
Paradoxical Abd movement		
Palpation		
Chest expansion		
Trachea		
Apex beat		
Tactile Vocal Fremitus (TVF)		
TVF on the lat wall		

Action		YES	NO
Percussion			
Ant	Percussion over the clavicles		
	Percussion Ant 2^{nd} to 6^{th} ICS		
	Percussion on the lat wall		
Post	Pt arms folded in front		
	Percussion on lung apices		
	No percussion on scapula or rib		
	Percussion on ≥4 sites		
Auscultation			
Ask the pt to breath form his mouth			
Warm the stethoscope			
Air entry			
Type of breathing			
Added sounds			
Vocal resonance			
In case of consolidation			
Whispered pectoriloquy			
Egophony			
Ethics			
Dress the patient back			
Thank the patient			

Respiratory system examination

Chest and Lungs	Student Comments/Questions
The following steps are performed with the patient sitting	AP diameter usually < transverse diameter, often by as much as ½
Inspection chest	**Barrel chest:** results from compromised respiration (chronic asthma, emphysema, cystic fibrosis). 1- Ribs more horizontal, 2- sternal angle more prominent.
✦ Size & shape (AP diameter compared with transverse diameter)	
✦ Scar and Deformity	**Pigeon chest**/pectus carinatum: prominent of sternum **Funnel chest**/pectus excavatum: indentation of lower sternum Substernal angle is obtuse in 1.Hyperinflation, 2.Kyphosis. Scar [surgical, old pleural tap]
✦ Superficial venous patterns	In Superior Vena Cava obstruction
Inspection chest movement with breathing:	
✦ Symmetry of chest expansion	↓ expansion in the diseased site
✦ Rhythm or Pattern	**Cheyne-stokes** (dying) – periods of ↑ depth interspersed with apnea **Kussmaul breathing** – rapid, deep, labored
✦ Paradoxical Abdominal movement	Is positive in pt with phrenic nerve paralysis
✦ Use of accessory muscles	Prominent Sternocleidomastoid and if the pt is assuming tripod position to use pectorlis major and minor muscles
Palpate the chest for the following :	
✦ Symmetry of Thoracic expansion	Done at 3 levels.from front or back to test all lung zones, it can be confirmed by a **tape** at level of 4th intercostal space TLC – RV (if <2cm is restricted).
✦ Mediastinal shift	Upper mediastinum by traceal deviation, Lower noted by apex beat.
✦ Sensations of transmitted sounds	**Crepitus:** crackly sensation; indicates air in subcut tissue (subcut emphysema) which may complicate pneumothorax **Pleural friction rub:** coarse vibration, usually on inspiration
✦ Tactile fremitus	**Tactile fremitus:** palpable vibration of chest wall that results from speech. **↓ or absent fremitus:** excess air in lungs,[emphysema, COPD and Asthma] Pleural thickening or effusion, Massive pulm edema, and Bronchial obstruction **↑ fremitus:** consolidation with patent bronchus Consolidation is presence of fluids or solid mass w/in lungs
Percussion	First: pt sitting w/head bent forward and arms folded in front Then ask pt to raise arms overhead to percuss lateral & anterior chest **Hyperresonance:** emphysema, pneumothorax. **Resonant:** Normal **Dullness:** consolidation & collapse = atelectasis, asthma **Stony Dull:** pleural effusion
Perform direct or indirect percussion on the chest, comparing sides: Note: (Apical lobe is behind the middle 1/3 of clavicle; percuss the axilla for middle lobe & lingula for Rt &Lt lung, respectively).	
✦ Site for percussion = Sites for auscultation	

Auscultation	Anteriorly: on the clavicle directly, and on the 2nd &4th intercostal space in midclavicular line and 6th intercostal space in anterior axillary line Laterally on the axilla Posteriorly: Apex + 3rd 6th & 9th Spaces
Auscultate the chest with the stethoscope diaphragm, from apex to base, comparing sides and comment for the following 4 items: ✦ Air entry ✦ Type of breathing. ✦ Added sounds (crackles, rhonchi, wheezes, friction rubs)	Normal, Reduced, or Absent. [Never ↑] Vesicular – continuous and inspiration > expiration Bronchovesicular – vesicular with prolonged expiration Bronchial - interrupted and inspiration = expiration Crackles (crepitations; rales) Rhonchi Pleural friction rub
✦ Vocal resonance	Whispered pectoriloquy: Whispering of sound [44]is heard as load sound in area of consolidation Egophony: whispering E is heard A– lung consolidation

Rhonchi definition: it's a musical sound due to airway obstruction and heard mainly during expiration and its 2 types:

1. Monophonic: its low pitched indicates local obstruction to major airway and so unilateral
2. Polyphonic: variable high pitched heard bilaterally indicated either asthma or COPD.

Crepitations

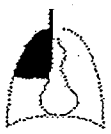
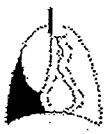
Type	Cause	Mechanism
Coarse = Harsh	Bronchiectasis	due to passage of air through bronchial secretions
Fine or Crackles	pulmonary edema	due to sudden opening of the collapsed alveoli at the end of the inspiration
Very fine and 'dry'	fibrosing alveolitis & IDL	

To differentiate between Bronchiectasis and ILD ask the patient to cough and if crepitations disappears its due to Bronchiectasis

Pleural rub -Diagnostic of pleurisy -> dry, rubbing sound with late inspiration and early expiration

If you hear bronchial breathing you should perform **Whispered pectoriloquy & Egophony** which occurs in Lung consolidation only.

Typical Chest Examination Findings					
Condition	Expansion	Percussion	Breath Sounds	TVF & Vocal resona	Adventitious Sounds
Normal	Normal	Resonant	Vesicular	Normal	Absent
Asthma	↓	Resonant	Vesicular	Normal or ↓	Wheezing
ILD	↓	Resonant	Vesicular	Normal	Fine Crepitations
Bronchiectasis	↓ or N	Resonant	Vesicular	Normal or ↓	Coarse Crepitation
Emphysema	↓	Hyperresonant	Decreased	Decreased	Wheezing
Ch. Bronchitis	↓ or N	Resonant	Vesicular	Normal or ↓	Crepitations & wheezing

	Consolidation	Pleural effusion	Lobar collapse	Pneumothorax
Chest xray				
Mediastinal shift	No	No or away	Towards	No (simple), away (tension)
Chest wall excursion	Normal or decreased	Decreased	Decreased	Normal or decreased
Percussion note	Normal or decreased	Decreased (stony)	Decreased	Increased
Breath sounds	Increased (bronchial)	Decreased	Decreased	Decreased
Added sounds	Crackles	Rub (occasional)	None	Click (occasional)
Tactile vocal fremitus/vocal resonance	Increased	Decreased	Decreased	Decreased

TVF is ↑ Bronchial breathing, whispering pectoriloquy, and Egophony occurs only in Consolidation or Collapse with patent bronchus.

Percussion note hyperresonance occurs only in Emphysema and Pneumothorax

TVF [Tactile vocal fremitus] comment on it when there is a difference between both sides of the lung otherwise sat its equal bilaterally

Important points in General examination:

Face:

- Look for pursed lip (COPD) and acting ala nasi
- Tongue for central cyanosis

Hand:

- Clubbing
- Nicotine staining
- Cyanosis
- CO_2 retention → rapid bounding Pulse, warm hands, and tremors

In the short case of pulmonary fibrosis or bronchiectasis:

1- Make sure that you ask the patient to cough, because if the case is bronchiectasis the coarse crepitations will disappear while the fine crepitations of pulmonary fibrosis will persist.
2- Please use tape measure to confirm the restriction of chest movement in patients of pulmonary fibrosis.
3- When finishing the chest examination of these cases the examiner will ask you "Do you like to examine any other thing in this patient?" Your answer will be "yes I like to examine the hands for clubbing.
4- In case of Bronchiectasis the examiner will tell you that after 20 years of this patient's disease the patient is started to develop renal failure and the examiner will ask what do think the cause is " your answer will be its renal failure due to Secondary Amyloidosis due to Bronchiectasis

Ix for Respiratory disease: For Any respiratory case say the following Ix:

Laboratory:

1- CBC, WBC Count, ESR & CRP
2- ABG

Radiology:

1- CXR
2- If bronchiectasis or CA lung in the DDx order CT scan
3- Bronchoalveolar lavage

Pulmonary Function Test

Differential diagnosis of clubbing		
Respiratory	**Cardiac**	**GIT**
Bronchial carcinoma	Cyanotic congenital heart	Cirrhosis
Lung abscess	disease	Ulcerative colitis
Empyema	Bacterial endocarditis	Crohn's disease
Bronchiectasis	Atrial myxoma	
Fibrosing alveolitis		**Familial**
Asbestosis	**Thyroid**	
	Grave's disease	

Test for clubbing:

- Fluctuation test
- Window test

Grades of clubbing:

- Grade I: Fluctuation (↑ ballotability)
- Grade II: Loss of the normal angle between the nailbed and the fold
- Grade III: Increased convexity of the nail fold (**Parrot's peak**)
- Grade IV: Finger looks like a **drumstick**

Asthma

Definition: Asthma is chronic inflammatory airway disease, with airway hyper responsiveness to a variety of stimuli, its **Episodic** (not continuous), its due to narrowing of the lower airway which is **Reversible** either spontaneously or by medication, and clinically by the **Triad** of dyspnea, cough, and wheeze

Clinical presentation

Symptoms: symptoms of asthma are the **triad** of dyspnea, wheeze, and cough.

Signs

- Vital signs: increased RR, increased HR, Pulsus Paradoxus
- Respiratory system examination:

A. Signs of respiratory distress:

1- Flaring of ala nasi
2- Use of accessory respiratory ms [sternocleidomastoid]
3- Suprasternal, Intercostal and Sucostal recessions.

B. by inspection the chest is hyperinflated with reduced chest expansion, there is no difference in tactile vocal fremitus, percussion note is resonant, no difference in vocal resonance, and in auscultation you here reduced air entry bilaterally with vesicular breathing, prolonged expiratory phase with expiratory rhonchi (may be both expiratory and inspiratory) with or without fine end-inspiratory crepitation.

Note: spontaneous pneumothorax rarely occurs.

Q. How to investigate a pt with asthma?

1- CXR: normal or shows Hyperinflation, or pneumothorax.
2- Pulse oxymetry: hypoxia, tachycardia.
3- ABG: (respiratory failure type I)

- O_2 → hypoxia
- CO_2 → reduced [if CO_2 is ↑ it is called life threatening asthma]
- pH → respiratory alkalosis

3- PFT: Obstructive pattern
4- PEFR

How to monitor Asthma?

By the use of **Peak Expiratory Flow Meter**

How to Diagnose Asthma?

- **Peak flow meter** → PEFR diary Greater than 20% diurnal variation on > 3 days in a week for 2 weeks {diurnal variation % = [(Highest – Lowest PEFR) / Highest PEFR] x 100}
- PFT will show Obstructive pattern (FEV1/FVC = < 70% of predicted)
  1. FEV1 > 15% after a short acting beta 2 agonist
  2. FEV1 > 15% after a 14 day course of prednisolone 30mg
  3. FEV1 > 15% decrease following 6 minutes of exercise

DDx: 1. Heart failure
2. GERD
3. COPD

Management

Management of asthma depends on the severity of the disease

Sever asthma	Life threatening asthma	Near fatal asthma
Defined as any of: • PEFR 33 to 50% • RR > 25 • HR > 110 • Inability to complete sentence in one breath • Pulsus paradoxus	Any one of: • PEFR < 33% • Silent chest, or Cyanosis , or Poor respiratory effort • Bradycardia or hypotension • Exhaustion, Confusion, Coma • ABG: low pH, N or ↑ CO_2, ↓ O_2	• Raised $PaCO_2$
Management of a pt with acute asthma (Status Asthmaticus): • ABC • **Oxygen: high concentration (>60%)** • **Bronchodilators: nebulised salbutamol** 2.5-5 mg repeat every 20 min if no improvement then add **ipratropium bromide** • **Steroids:** oral prednisolone 40 mg or IV hydrocortisone 200 mg is added after the 3rd dose of nebulised salbutamol if there is no improvement. Steroid is continued for 5 days. • Magnesium sulphate for patients who are not responding single dose only – if no response → IV aminophylline if no response consider IV salbutamol. If no response • Do CXR to exclude pneumothorax • Sedation must be avoided		Management admission to ICU for incubation and mechanical ventilation

What are the side effects of β-agonists?
- Tachycardia
- Irritability

How long it take corticosteroids to act in acute asthma?
It takes about 6 hours.

If the pt is not improving on treatment what are the possibilities?
- The pt may have developed pneumothorax
- The Oxygen pump is empty

History of Asthma = History of pts in which the main complain is wheeze

MC: Wheeze

Examiner must determine the following:

1-Onset: At what age did the wheezing begin?

2-Frequency: of day symptoms, night symptoms, and exacerbations\year,

3-Progression: Have the symptoms worsened over the years?

4-Duration:

5-Diurnal variation:

6-Severity: effect on sleep? And rest of asthma effects on pt are covered in SH

7-Are there any precipitating factors, 1.odors, 2.any type of food, 3.animals, 4.cold air, 5.URTI, 6.occupation, 7.drugs, 8.emotions, 9 Exercise.?

8-What usually stops the attack?

9-Are there any associated symptoms?

10-Is there a history of allergic rhinitis and nasal polyps? [Remember the syndrome]

11-Is it seasonal or perennial?

["What is your smoking history?" "Is there a history of heart disease?" "Is there Sx of heartburn?"]

All of these questions are for DDx in adult pt.

Aspirin-Sensitive Asthma *"Samter's Triad"* or *"Widal's Syndrome"*

1- Rhinosinusitis

2- Recurrent Nasal polyps [ask about them in Hx]

3- Asthma that is exacerbated by aspirin

Note that Q number 1, 7, 10, 11 will help distinguishing if the pt disease is intrinsic or extrinsic

Ask about other respiratory symptoms

Note: Asthmatic pt may have productive cough which is usually white in color but occasionally they develop Yellowish sputum but this not necessarily mean infection because the yellow color may be due to eiosinophilia.

ROS: GIT and CVS are important

PMH: when did the first attack occur and in adult pt ask specifically if he/she had the disease when he was a child

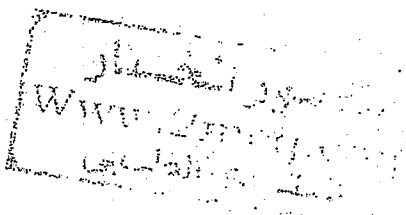
Ask about the number of previous admissions due to asthma

Admission to an ICU

Drugs used and their side effects.

FH: for the Atopy group

SH: MOST IMPORTANT (Ask about exposure to smoke, Ask about house ventilation, Exposure to pets, going to farm. Sport related activity, School Performance, Days missed from work.



Chronic Obstructive airway disease

Definitions

Chronic bronchitis (clinical definition): it is the presence of productive cough for at least 3 months in a year for 2 successive years.

Emphysema (pathological definition): it is abnormal permanent enlargement of the airway distal to the terminal respiratory bronchioles.

Epidemiology: it is the 4th leading cause of death in the world

Etiology: Smoking [15% of smokers will develop COPD]

	Emphysema (Pink puffer)	Chronic bronchitis (Blue bloater)
Built	Thin	Obese
Cyanosis	Absent	Prominent
Dyspnea	++	+
Hyperinflation	+++	+
Cor pulmonale	rarely	often

Clinical picture

Symptoms

- Dyspnea
- Productive cough
- Wheeze

Signs

- ↑ RR
- Hyperexpanded/barrel chest with pursed lip breathing
- Look for nicotine stain
- Quiet breath sounds (especially in the lung apices) ± wheeze
- Quiet heart sounds ± impalpable apex beat (due to overlying hyperinflated lung)
- Mild inspiratory crackles

The patients may develop symptoms of respiratory failure type II:

- ↑ CO_2 → vasodilation leading to:
  1. ↑ ICP → headache and papilledema
  2. Large volume pounding pulse
  3. Warm hands & palmar erythema
- Ch ↑ in CO_2 → polycythemia
- Asterixis = flapping tremor

DDx of flapping tremors:
- Respiratory failure
- Liver failure
- Cardiac failure
- Renal failure

Q: What is the disease in which the pt has emphysema and liver cirrhosis?

α1- Antitrypsin deficiency

Investigation

- CXR will show hyperinflated lung fields:
  1. More than 7 posterior ribs seen
  2. Flattened diaphragms
  3. More horizontal ribs

Note: lung bullae may be seen, which if large can be mistaken for a pneumothorax due to the loss of lung markings (CT can differentiate).

- ABG: may show respiratory failure type II
- Pulse oxymetry
- PFT: Reduced FEV_1 to <80% predicted and there is Minimal bronchodilator reversibility (<15%, usually <10%).
- For Emphysema the best diagnostic method is CT scan.

Management

Treatments that ↓ mortality (Smoking cessation and Oxygen therapy)

Treatment of acute exacerbation of COPD:
- *Bronchodilators:* Short-acting β-agonists (salbutamol) ± anticholinergics (ipatroprium)
- *Systemic Glucocorticoids*
- **Antibiotics** if sputum purulent, pyrexial, ↑CRP, new changes on CXR.
-

> - Physiologically CO_2↑ in or ↓O_2 can stimulate the respiratory center
> - In normal person the blood level of CO_2 drives respiration
> - In pts with COPD **chronic** elevation of CO_2 results in tolerance of the respiratory center to CO_2 and the respiratory center will depend on a ↓ O_2 (hypoxia) to stimulate respiration; This is termed **Hypoxic Drive**, therefore in pts with COPD high dose oxygen can reduce respiration and cause respiratory depression which may require mechanical ventilation.

Controlled Oxygen therapy with concentration of **24to28% via Venturi mask.**
- **Doxapram:** Intravenous respiratory stimulant drug. Can be used to drive respiratory rate (if below 20 per minute)
- ***Ventilatory Support*** if pt didn't improve

Bronchiectasis

Definition: Irreversible abnormal dilatation of one or more bronchi, with chronic airway inflammation and obstruction.

Causes
- TB
- Post-pneumonia
- Cystic fibrosis
- Kartagner's syndrome (Bronchiectasis, dextrocardia, ch sinusitis ± situs inversus)
- Yellow nail syndrome
- Idiopathic

Clinical picture
- Symptoms
  - Cough
  - Chronic production of large amount of highly viscous purulent sputum especially on the morning
  - Intermittent hemoptysis
  - Breathlessness,Lethargy/malaise.
- Signs
  - Coarse inspiratory and expiratory crackles on auscultation
  - Airflow obstruction with wheeze.

> Complications:
> - Respiratory failure
> - Recurrent infection
> - Pulm HT
> - Amyloidosis (AA type) result in nephrotic syndrome

Dx: High resolution CT

Rx: • Bronchodilator for obstruction • Antibiotics for infections
• Mucolytics for thick sputum

Interstitial lung diseases (ILD)

Definition

Interstitial lung disease (ILD)--or pulmonary fibrosis--refers to chronic lung disorders of both known and unknown etiology that are characterized by involvement of the tissue between the air sacs of the lungs (**the interstitium**) which is affected by inflammation or scarring (fibrosis of lung parenchyma).

So why all of these diseases are both under the single heading?
This is because:

- All of them share similar Pathophysiology which is the involvement of the alveolo-capillary membrane (Blood-Air barrier) i.e. they involve Gas-Exchange.
- This is because all of these heterogeneous groups of disorders share *similar clinical, roentgenographic, physiologic, & pathologic manifestations.* BUT a specific diagnosis should be obtained in all cases because the causes of ILD differ in their treatment and their prognosis.

Classification

Lung Response: Alveolitis, Interstitial Inflammation, and Fibrosis	
Known Cause	Unknown Cause
1. Asbestosis 2. Radiation 3. Drugs (Nitrofurantoin, Amiodarone, Gold) and chemotherapy drugs	1. Idiopathic pulmonary fibrosis = Cryptogenic fibrosing alveolitis 2. Connective tissue diseases (SLE, RA, S 3. Amyloidosis

Lung Response: Granulomatous	
Known Cause	Unknown Cause
Hypersensitivity pneumonitis (organic dusts) Pneumoconiosis (Inorganic dusts: silicosis)	Sarcoidosis

Clinical picture

History

Main complains are progressive dyspnea (usually Exertional) and Dry cough.
History is very important as it may reveal the underlying cause for example:
Past medical history → collagen vascular disorder
Social history → exposure to pets, & smoking
Occupational history → exposure to organic or inorganic dusts
Drug history → drugs may be the underlying cause

Examination

General examination may show evidence of systemic disease (Sarcoidosis or collagen vascular diseases); examination may show complication of ILD→ PHT and RVF.

Respiratory examination

Inspection→ reduced chest expansion bilaterally
Palpation→reduced chest expansion can now be confirmed by the use of Tape measure placed at the 4th intercostal space and difference in chest diameter between full inspiration and full expiration is measured [if < 2 cm it's abnormal]

Auscultation → Fine end-inspiratory crepitations that does not disappear after asking the patient to cough (the mechanism of crepitations here is Sudden reopening of the collapsed alveoli)

Investigations

Laboratory test

- CBC→ lymphopenia in sarcoidosis; eosinophilia in pulmonary eosinophilias and drug reactions; neutrophilia in hypersensitivity pneumonitis.
- CRP and ESR
- Rheumatoid factor & Antinuclear antibody
- Ca^{2+} level may increased in Sarcoidosis

Imaging

- CXR
- High-resolution CT (HRCT) is the imaging investigation of choice because more sensitive and specific, CXR & HRCT changes include:
    1. reticular (crisscrossing lines)
    2. nodular (lots of small dots)
    3. rings or cysts (honeycombing in advanced disease)
    4. ground glass

PFT

- Spirometery→ will show restrictive pattern (FEV1/FVC is > 70%) but TLC and RV are reduced
- Carbon Monoxide diffusion capacity (DL_{CO}): in this test, a small concentration of carbon monoxide (0.3%) is inhaled, usually in a single breath that is held for ~10 The value obtained for DL_{CO} depends on
    1. The alveolar-capillary surface area→ ↓ in emphysema
    2. Pulmonary capillary blood volume → ↓ in pulmonary embolism
    3. Thickness of the alveolar-capillary membrane → ↓ in ILD
    4. The patient's hemoglobin level will affect the measurement.

Note: The main use for DL_{CO} is in the investigation for ILD as it's the most sensitive functional test.

Fibrosis mainly affecting the upper zones	Fibrosis mainly affecting the lower zones
1. Extrinsic allergic alveolitis	1. Cryptogenic fibrosing alveolitis
2. Silicosis	2. CTD (except ankylosing spondylitis)
3. Ankylosing spondylitis (rare)	3. Drug-induced
4. Tuberculosis	4. Asbestosis

BAL (bronchoalveolar lavage)

Disease	Finding on BAL
Idiopathic pulmonary fibrosis	Neutrophilia
Sarcoidosis	Lymphocytosis with CD4 predominance
Hypersensitivity pneumonitis	Lymphocytosis with CD8 predominance

Biopsy

Lung biopsy is required when the diagnosis of interstitial lung disease is not clear and it may be transbronchial or open lung biopsy.

Lung biopsy is the gold standard for making the diagnosis of ILD.

Pathophysiology of ILD

Fibrosis of the lung → reduced compliance → ↑ work of breathing → dyspnea

Thickening of the alveolo-capillary membrane → limitation of **oxygen diffusion** mainly on exercise but not at rest unless the disease is so sever.
Hypoxia in the lung → vasoconstriction of pulmonary blood vessels –by time→ Pulmonary HT → right ventricular hypertrophy –by time→ RVF.

Cyanosis occur at end-stage of the disease

Why this limitation of diffusion is applied on O_2 but not on CO_2?
CO2 usually does not go up because it is 20 times more diffusible than oxygen

ILD → TYPE I RESPIRATORY FAILURE

Idiopathic pulmonary fibrosis = Cryptogenic fibrosing alveolitis

Clinical picture
History
- It's a disease of elderly > 50 years, more common in males; rarely familial.
- Gradual onset of dyspnea (especially on effort) and dry coughing often accompanied by constitutional symptoms (fatigue, anorexia, weight loss).
- One-third of pts date symptoms to after a viral respiratory infection (EBV).
- Smoking history is common.

Physical Exam
- End-inspiratory crackles at posterior lung bases.
- **Clubbing** is common.

Laboratory Findings
- ESR and LDH may be elevated.
- Rheumatoid factor and ANA are found in 30-50% of patients.
- Hypoxemia is common, but polycythemia is rare.

Imaging Studies
- CXR: Bilateral basal reticulonodular markings ± honeycombing.
- High-resolution CT is the best imaging modality because it show abnormalities when CXR is normal (i.e. early in the disease); HRCT may be diagnostic.

PFTs: Typically restrictive pattern with reduced total lung capacity. DLCO often decreased; mild hypoxemia, which worsens with exercise.
BAL: Neutrophilia ± eosinophils
Histologic Findings: biopsy confirms the diagnosis and is very important in predicting response to treatment
Treatment
Corticosteroid and immunosuppressive (no treatment can improve prognosis)
Prognosis median survival of 3 years is typical and survival beyond 5 years unusual.
Note: there is rare acute form (**Hamman-Rich syndrome**) occurs with death within months

Abdominal Examination checklist

Action	YES	NO	Action	YES	NO
Permission taken			Percuss for liver span		
Stood on the Right side of bed			Palpate for spleen		
Adequate Exposure Position the patient			Palpate spleen in Rt lat position Palpate for renal enlargement		
Inspection			Check hernial orifices		
Stand at the end of the bed			**Percussion**		
Symmetrical or not			Shifting dullness		
Swelling or distention			Fluid thrill		
Movement with respiration			**Auscultation**		
Scar			Warm stethoscope		
Stria			For Intestinal movement		
Superficial veins			For bruit if hepatomegaly		
Umbilicus			**I would like to**		
Palpation			I would like to examine genitalia and do DRE.		
Ask the pt if there is pain in abd					
Kneel down + Warm hands			**Ethics**		
Do superficial palpation			Dress the patient back		
Do deep palpation			Thank the patient		
Eyes kept on pt face					
Palpate for the liver					

Abdominal Examination

Preparation

- Permission
- Ensure the patient is lying flat (remove any extra pillows, if' present, with the permission of the patient); the hands should lie by the patient's side with the
- Abdomen exposed from the inframammary region to just above the genitalia. Do not expose the genitalia.

Inspection

Scar: Midline, Suprapubic, Lt subcostal, Laparoscopy
Distention: Fat, Fluid, Flatus, Feces, Fetus
Veins: Caput Medusae (portal hypertension) Lateral veins (SVC or IVC), (check direction of flow, which is usually away from the umbilicus).

Difference between Ascites and Fatty distension	
Ascites	Fatty distension
Distension of the flanks Umbilicus flat or everted	Distension is central Umbilicus is inverted

Ascites
Minimal → Dullness only in Knee-elbow position
Moderate → Shifting dullness positive
Tense (severe) → Transmitted thrill

Palpation

- The patient should be asked to place arms at the side of the body
- Ask about the site of the pain
- The patient face should be observed for any expression
- In superficial palpation the examiner's hand should remain in continuous contact with the patient's abdomen
- When palpating for organomegaly never take off your hand until you finish

Superficial Palpation:

1- to be familiar with the patient
2- Tenderness
3- Rigidity (involuntary ms cont)
4- Guarding (voluntary ms cont)

Deep Palpation:

1- Liver
2- Spleen
3- Kidneys
4- Masses

When liver is palpable how to differentiate between hepatomegaly and ptosed liver?
By the liver span (Normal liver span is <12cm)

Causes of hepatomegaly:

1- Heart failure
2- Liver cirrhosis
3- Malignancy [leukemias, primaries, or secondaries]
4- Infections: glandular fever, infectious hepatitis, and hydatid disease

Enlarged liver: comment on its size, tenderness, surface (smooth or irregular), and consistency; percuss the upper border (normally in the fifth intercostal space in the right midclavicular line) and auscultate for bruit.

- Tender liver indicates a stretch of its capsule (Glisson's capsule) due to a recent enlargement, as in cardiac failure or acute hepatitis.
- Pulsatile liver → Tricuspid regurgitation.
- Hepatic arterial bruit over the liver indicate → alcoholic hepatitis, primary or metastatic carcinoma.
- Abdominal venous hum indicate → diagnostic of portal venous hypertension

Murphy sign: pain on palpation of the right subcostal area during inspiration frequently associated with acute cholecystitis.

Spleen

Spleen lies under 9th, 10th, & 11th rib with anterior margin reaching anterior axillary line

Sizes:

Spleen is palpable if it's 3 times more enlarged than normal

Massive Splenomegaly is >8cm below costal margin or crosses midline

Not every palpable liver is pathological but any palpable spleen is pathological

Methods to palpate for the spleen:

1- Normal starting in the Rt iliac fossa
2- Short's maneuver (bimanual exam in Rt lat position)
3- Percussion on Traube's area (a crescentic space about 12 cm wide, bounded medially by the left border of the sternum, above by an oblique line from the 6th costal cartilage to the lower border of the 9th rib in the midaxillary line and below by the costal margin; the percussion tone here is normally tympanitic, because of the underlying stomach, but is dull in presence of enlarged spleen).

Splenomegaly

- Spleen lies under 9th, 10th, & 11th rib with anterior margin reaching anterior axillary line
- Sizes: Spleen is palpable if it's 3 times more enlarged than normal
- Massive Splenomegaly is >8cm below costal margin or crosses midline
- Not every palpable liver is pathological but any palpable spleen is pathological

Massive splenomegaly >8cm	Moderate splenomegaly 4-8c	Slight < 4cm
1- Myelofibrosis	Causes of Massive and:	Causes of Massive & Moderate an
2- CML	1- Hemolytic anemia	1- Infections [IMN, SBE]
3- Malaria	2- Lymphoproliferative dis	2- Blood dis [PRV, ITP, pernicious
4- Kala-azar	3- Portal hypertension	anemia, SLE, Felty's. Sarcoidosis,
5- Gaucher's disease	4- Splenic vein thrombosis	Amyloidosis]

- *Hypersplenism*:
  - *Definition:* its condition characterized by splenomegaly + cytopenia(s) + hyperplastic bone marrow + a response to splenectomy.
  - It occurs as a result of that components of the blood (RBC, WBC, Plt) are removed at an abnormally high rate by the spleen → low circulating levels.

Differences between Splenic and Renal mass		
	Spleen	Renal
1-	Can not get above it	Can get above
2-	Move downward and medially with respiration	No
3-	Notch may be felt	No
4-	No	Ballotable
5-	Dull on percussion	Resonant on percussion

Causes of hepatosplenomegaly:

1- Infections→ Chronic hepatitis, IMN, CMV
2- Malignancy → leukemia, lymphoma
3- Extramedullary hematopoisis

Masses

Right iliac fossa mass	Left iliac fossa mass
1- Carcinoma cecum 2- Crohn's disease 3- Appenicular mass 4- Iliocecal TB 5- Iliac lymphadenopathy 6- Pelvic or transplanted kidney	1- Carcinoma of colon 2- Diverticular 3- Iliac lymphadenopathy 4- Pelvic or transplanted kidney

Epigastric mass	Suprapubic mass
1- Gastric CA 2- Pancreatic pseudocyst 3- Aortic aneurysm (expansile pulsation)	1- Bladder 2- Uterus 3- Ovarian cyst

Auscultation for:

1- Arterial bruit in renal artery stenosis
2- Venous hum in portal hypertension
3- Intestinal sound

Liver Cirrhosis [Causes, Complications and their treatment]

Definition

Chronic disease of the liver characterized by fibrosis, disorganization of the lobular and vascular architecture, and regenerating nodules of hepatocytes.

Cirrhosis = Fibrosis + Disorganization of structure + Regeneration nodule

Causes

- Alcoholic liver disease 60% to 70% the most common cause
- Viral hepatitis (B, C, D)10%
- Biliary diseases 5% to 10%
- Primary hemochromatosis 5%
- Wilson disease Rare
- α_1-Antitrypsin deficiency Rare
- CHF (cardiac cirrhosis)
- Cryptogenic cirrhosis 10% to 15%

Clinical presentation

Symptoms

Anorexia, nausea, vomiting, diarrhea, fatigue, weakness, fever, jaundice, amenorrhea, impotence, & infertility

Signs

Signs of Chronic liver disease in GE	
Site	Signs
Hands	Clubbing, Leuconychia, Palmar Erythema, Dupuytren's contracture, Spider naevi [Spider naevi of > 5 is pathological], Liver flap
Face	Spider naevi, Telangiectasia, Jaundice, Pigmentation, Central cyanosis
The limbs	Ankle edema
Trunk	Gynecomastia, Excoriation

Laboratory Findings **Diagnostic test**

CBC: Anemia (microcytic due to blood loss, macrocytic due to folate deficiency), pancytopenia (hypersplenism). **RBS:** hypoglycemia.

Coagulation test: prolonged PT, rarely overt DIC.

Urea and electrolyte: hyponatremia.

ABG: hypokalemic alkalosis, hypoxemia (hepatopulmonary syndrome).

Blood protein: hypoalbuminemia (albumin level < 3.5 g).

Complications

1. Portal hypertension
2. Ascites
3. Spontaneous peritonitis
4. Esophageal varices
5. Splenomegaly
6. Hepatic encephalopathy
7. Hepatorenal syndrome
8. Hepatopulmonary syndrome
9. Bleeding tendency
10. Hepatocellular carcinoma

Portal hypertension

Definition: Portal hypertension is defined as an increase in portal vein pressure (>10 mmHg) due to anatomic or functional obstruction to blood flow in the portal system.
Normal portal vein pressure is 5–10 mmHg.

Pathophysiology

Increased portal pressure will result in the following:

1. Increased collateral circulation between high pressure portal venous system & low-pressure systemic venous system (in sites of portosystemic shunts)will cause:
    - lower esophagus/upper stomach (varices, portal hypertensive gastropathy),
    - rectum (varices, portal hypertensive colopathy),
    - anterior abdominal wall (caput medusae; flow away from umbilicus)
    - note: portosystemic shunting is a risk factor for hepatic encephalopathy

2. Ascites
3. Splenomegaly ± hypersplenism

Causes of portal hypertension	
Site of obstruction	
Presinusoidal	Splenic AV fistula, portal or splenic vein thrombosis, schistosomiasis
Sinusoidal	Cirrhosis, hepatitis
Postsinusoidal	Budd-Chiari syndrome, venoocclusive disease

Budd-Chiari syndrome, or hepatic vein thrombosis, is associated with hypercoagulable states, pregnancy, tumors, abdominal trauma.

Hepatic Encephalopathy

Definition: A state of disordered CNS function associated with severe acute or chronic liver disease; may be acute and reversible or chronic and progressive. Asterixis (flapping tremor) are present unless the patient is unconscious
There is a characteristic EEG abnormality.

Clinical presentation
Stage 1: euphoria or depression, mild confusion, slurred speech, disordered sleep,
Stage 2: lethargy, moderate confusion.
Stage 3: marked confusion, sleeping but arousable, inarticulate speech.
Stage 4: coma; initially responsive to noxious stimuli, later unresponsive.

Pathophysiology

- Failure of liver to detoxify agents noxious to CNS, i.e., ammonia, mercaptans, fatty acids, γ-aminobutyric acid (GABA), due to decreased hepatic function and portosystemic shunting.
- Ammonia may deplete brain of glutamate (excitatory neurotransmitter).
- Endogenous benzodiazepine agonists may play a role.
- Blood ammonia most readily measured marker, although may not always correlate with clinical status.

Precipitant factors

1. GI bleeding (100 mL = 14–20 g of protein),
2. Azotemia
3. Constipation
4. High-protein meal
5. CNS depressant drugs (e.g., benzodiazepines)
6. Hypoxia, Hypercarbia, Sepsis

Treatment

- Remove precipitants; reduce blood ammonia by decreasing protein intake and enemas/cathartics to clear gut.
- Lactulose oral (converts NH3 to unabsorbed NH4, produces diarrhea, alters bowel flora). In coma, give as enema.
- In refractory cases, add neomycin, metronidazole, or vancomycin.
- Flumazenil, a short-acting benzodiazepine receptor antagonist, is used if encephalopathy is precipitated by benzodiazepine use.
- If all fail then Liver transplantation is indicated.

Esophageal varices

Bleeding is major life-threatening complication; risk correlates with variceal size and the degree of portal hypertension (portal venous pressure > 12 mmHg).

Mortality correlates with severity of underlying liver disease (hepatic reserve), and correlates with Child-Pugh criteria

Child-Pugh Classification of Cirrhosis				
Factor	Units	1	2	3
Serum bilirubin	mol/L	< 35	35-51	>51
Serum albumin	g/L	> 35	30-35	<30
Prothrombin time	seconds	<4	4-6	>6
Ascites		None	Controlled	Poorly controlled
Hepatic encephalopathy		None	Minimal	advanced

Child-Pugh classification with a scoring system of 5–15:

- Class A (score of ≤ 6): consistent with compensated cirrhosis.
- Class B (scores of 7–9): decompensated cirrhosis.
- Class C (scores of 10–15): end-stage liver disease

Uses of Child-Pugh:

1. Reliable predictor of survival in liver diseases and predicts the likelihood of major complications of cirrhosis such as bleeding from varices and spontaneous bacterial peritonitis.
2. It was used to assess prognosis in cirrhosis
3. Provide the standard criteria for listing for liver transplantation (class B)

Investigation

Esophagogastroscopy: procedure of choice for evaluation of upper gastrointestinal hemorrhage in pts with known or suspected portal hypertension.

Treatment

Primary prevention of variceal bleeding

- **propranolol** (portal venous antihypertensives) - the best current treatment
- nitrates
- endoscopic banding for large varices
- note sclerotherapy has no real role in primary prevention

Treatment of variceal hemorrhage

- ABC
- Correct clotting: FFP, vitamin K

1. Endoscopic band ligation or sclerotherapy: **procedure of choice**; banding is superior to sclerotherapy.
2. Medical treatment [used only in active bleeding]
   a. IV octreotide (Somatostatin analog)
   b. Vasopressin (terlipressin) used with add nitroglycerin to prevent coronary and renal vasoconstriction
3. Sengstaken-Blakemore tube if uncontrolled hemorrhage
4. Transjugular Intrahepatic Portosystemic Shunt (TIPS) if above measures fail

Prevention of recurrence

- Propranolol
- Banding
- Sclerotherapy
- TIPS

ASCITES

Definition

Accumulation of fluid within the peritoneal cavity.

Causes (DDx)

1. Cirrhosis
2. CHF
3. Nephrotic syndrome
4. Tuberculosis
5. Malignancy
6. Myxedema
7. Others Infections (bacterial,tuberculous, fungal, parasitic),CTD, Pancreatitis, Whipple's disease, familial Mediterranean fever, Budd-Chiari syndrome.

Symptom

- Small amounts may be asymptomatic.
- Large amounts cause abdominal distention and discomfort, anorexia, nausea, early satiety, heartburn, flank pain, and respiratory distress.

Examination

Fullness of flanks, umbilicus flat or everted

Difference between Ascites and Fatty distension	
Ascites	Fatty distension
Distension of the flanks	Distension is central
Umbilicus flat or everted	Umbilicus is inverted

Degree of ascites

Minimal → Dullness only in Knee-elbow position

Moderate → Shifting dullness positive

Tense (severe) → Transmitted thrill

Investigations

Ultrasound: Very sensitive; able to distinguish fluid from cystic masses.

Diagnostic paracentesis (50–100 mL): Routine evaluation includes inspection, albumin, glucose, cell count and differential, Gram's and acid-fast stains, culture, cytology; in selected cases check amylase, LDH, triglycerides, culture for TB. Rarely, laparoscopy or even exploratory laparotomy may be required.

Pathophysiological classification using serum ascites albumin gradient

Difference in albumin concentrations between serum and ascites as a reflection of imbalances in hydrostatic pressures:

1. *Low gradient* (serum-ascites albumin gradient <1.1): bacterial peritonitis, neoplasm, pancreatitis, nephrotic syndrome.

2. *High gradient* (serum-ascites albumin gradient > 1.1 suggests ascites is due to portal hypertension): cirrhosis, CHF, Budd-Chiari syndrome.

Pathophysiology of cirrhotic ascites:

1. Portal hypertension
2. Hypoalbuminemia
3. Hepatic lymph
4. Renal sodium retention—secondary to hyperaldosteronism, increased sympathetic nervous activity (renin-angiotensin production).

Treatment

Maximum mobilization <700 mL/d (peripheral edema may be mobilized faster).

1. Rigid salt restriction (400 mg Na/d).
2. Fluid restriction of 1–1.5 L only if hyponatremia.
3. Diuretics: spironolactone (mild, potassium-sparing, aldosterone-antagonist) furosemide may be added if necessary.
4. Monitor weight,urinary Na and K, serum electrolytes, and creatinine.
5. Repeated large-volume paracentesis (5 L) with IV infusions of albumin (10 g/L ascites removed) is preferable for initial management of massive ascites because of fewer side effects than diuretics.
6. In refractory cases, consider transjugular intrahepatic portosystemic shunt (TIPS), though 20–30% risk of encephalopathy and high rate of shunt stenosis and occlusion. Consider liver transplantation in appropriate candidates

Rheumatoid Arthritis

Definition

Rheumatoid arthritis is a chronic immunologically mediated inflammatory disorder of unknown cause that is characterized by synovial cell proliferation and inflammation with subsequent destruction of adjacent articular tissue, therefore cartilaginous destruction, bony erosions, and joint deformity are hallmarks of RA. The presentation is by polyarticular, symmetrical joint involvement, ± extra-articular involvement.

Epidemiology

Prevalence: worldwide prevalence is **1%.**
Age: peak age of onset is early 40s.
Gender: it is more common in **women** with F: M ratio = **3**:1. But the sex ratio varies with age (at 30 years F/M is 10:1, at 65 years 1:1).
HLA associations: HLA-DR4.

Etiology & Pathogenesis

Unknown antigen → Immune mediated response → inflammation → Synovial tissue proliferation (Pannus) → destructive arthropathy.

Clinical manifestations

Articular manifestations—typically a symmetric polyarthritis of peripheral joints with pain, tenderness, and swelling of affected joints; morning stiffness is common; it characteristically involves hand joint (PIP, MCP, wrist) and feet joints (MTP) and knees; joint deformities may develop after persistent inflammation.
PIP: proximal interphalangeal, MCP: metacarpophalangeal, MTP: metatarsophalangeal
Extraarticular manifestations:
Cutaneous—rheumatoid nodules, vasculitis, palmar erythema, Raynaud's syndrome and pyoderma gangrenosum.
Pulmonary— pleural effusion is the most common pulmonary complication, more in male, nodules, interstitial disease, bronchiolitis obliterans–organizing pneumonia (BOOP), Caplan's syndrome [sero(+) RA associated with pneumoconiosis]
Ocular-- common, with 25% of patients having eye problems, keratoconjunctivitis sicca is the most common, episcleritis, scleritis, and scleromalacia perforans.
Hematologic—anemia, Felty's syndrome (splenomegaly and neutropenia)
Cardiac—pericarditis (most common cardiac complication), myocarditis
Neurologic—myelopathies secondary to cervical spine disease, entrapment syndromes e.g. carpal tunnel syndrome, vasculitis causing mononeuritis multiplex, and peripheral neuropathy with 'glove and stocking' distribution.
Others—Amyloidosis.

Examination of rheumatoid hands					
Action	YES	NO	**Action**	YES	NO
Permission taken			**Range & Power**		
Stood on the Right side of bed			Wrist		
Adequate Exposure			Abduction		
Ask pt to put hands on the pillow			Adduction with paper test		
Inspection			Thumb to Fingers opposition		
Nails			Hand grip		
Skin (Scar, Palmar erythema)			**Functional assessment**		
Ms (Wasting)			Unbuttoning & buttoning		
Joints (Deformity & Swelling)			Writing		
Palpation			**I would like to**		
Ask if there is any pain			Look for other joints, splenomegaly and lower lobe fibrosis.		
Hotness					
Tenderness			**Ethics**		
For Area of swelling			Dress the patient back		
For Rheumatoid nodules			Thank the patient		

What are the typical changes of RA in hands?
(1) Radial deviation at the wrist with ulnar deviation of the digits, often with palmar subluxation of the proximal phalanges.
(2) Hyperextension of the proximal interphalangeal joints, with compensatory flexion of the distal interphalangeal joints **(Swan-neck deformity).**
(3) Flexion contracture of the proximal interphalangeal joints and extension of the distal interphalangeal joints **(Boutonnière deformity)**
(4) Thumb: Hyperextension of the interphalangeal joint and flexion of the first metacarpophalangeal joint with a consequent loss of thumb mobility and pinch. **("Z" deformity)**

What determines if the disease is active or not = inflamed or not?
Presence of hotness, tenderness ± rheumatoid nodules

How to differentiate between Acute & Chronic RA?
The presence of joint deformities and muscle wasting indicates chronic disease

What precautions are necessary before upper gastrointestinal endoscopy or general anesthesia in patient with RA?
It is prudent to take a cervical spine radiograph to rule out atlanta-axial subluxation.

In patient with RA who develops proteinuria how can you explain that?
The kidney in RA may be affected by:

- Amyloidosis resulting in nephrotic syndrome.
- Drug therapy: gold salts, penicillamine, both can cause nephrotic syndrome.

How the eye becomes involved in RA?

- Keratoconjunctivitis sicca due to secondary Sjögren's syndrome is (most common ocular manifestation RA pts), it is characterised by dry, burning and gritty eyes due to decreased tear production
- Episcleritis (erythema)
- Scleritis (erythema and pain)
- Corneal ulceration
- Keratitis.
- Scleromalacia.
- Scleromalacia perforans.
- Iatrogenic causes of eye disease in pts with RA
  a. steroid-induced cataracts
  b. chloroquine retinopathy

*What do you about the **rheumatoid nodules**?*

- They are present in about 25% of cases.
- Site: On extensor surface of ulna and over the olecranon, flexor and extensor tendons of the hand, sacrum, Achilles tendon, sclera, lungs, pleura, myocardium, & meninges.
- They are usually asymptomatic
- They are one of the criteria for diagnosis of RA
- There presence indicates:
  1- Active disease
  2- RF is being + in 100%
  3- More aggressive disease with extra-articular manifestations

*What is **Felty's syndrome**?*

- Felty's syndrome consists of chronic RA, splenomegaly, neutropenia ± anemia & thrombocytopenia, lymphadenopathy and hepatomegaly are common.
- It is more common in females, and occurs in old age > 50 years.
- Splenectomy ameliorates hypersplenism.
- Felty's syndrome is associated with positive rheumatoid factor & rheumatoid nodules

What factors have been implicated in anemia of chronic disease?

1- Decreased production of red blood cells
2- Due to inadequate iron: impaired absorption and transport, failure to release iron stores. [**Hepcidin** related]
3- Due to decreased concentration or marrow resistance to erythropoietin.
4- Increased destruction of red cells.

Investigations

- Rheumatoid factor:
  - Rheumatoid factor (RF) is a circulating antibody (usually IgM) which reacts with antigenic sites on the Fc portion of the patients own IgG
  - Is present in 85% of pts; its presence correlates with severe disease, nodules, and extraarticular features, (but NOT a marker of disease activity)
- CBC:
  - RBC → normocytic normochromic anemia.
  - WBC → Leukocytosis or neutropenia (Felty's syndrome).
  - Platelets →Thrombocytosis or thrombocytopenia (Felty's syndrome).
  - ESR and CRP: both are elevated.
- Synovial fluid analysis—useful to rule out crystalline disease, infection.
- Radiographs—**x-ray changes** include:
  - Early x-ray findings
    - loss of joint space, i.e. narrowing of joint space.
    - juxta-articular osteoporosis = periarticular osteopenia.
    - soft-tissue swelling
  - Late x-ray findings
    - periarticular erosions, also called marginal erosions.
    - subluxation
- CXR should be obtained.

Monitoring

ESR, CRP and hemoglobin levels are used to monitor response to treatment.

Diagnosis: Diagnosis is based on diagnostic criteria

Diagnostic criteria for the diagnosis of RA

	Criterion	Definition
1	Morning stiffness	Morning stiffness in and around the joints, lasting at least 1 hour before maximal improvement
2	Arthritis of three or more joints	Soft tissue swelling or fluid (not bony overgrowth alone) observed by a physician. 14 possible areas are right or left PIP, MCP, wrist, elbow, knee, ankle, and MTP joints.
3	Arthritis of hand joints	At least one area swollen in a wrist, MCP, or PIP joint
4	Symmetrical arthritis	Simultaneous involvement of the same joint areas bilaterally (bilateral involvement of PIPs, MCPs, or MTPs is acceptable without absolute symmetry)
5	Rheumatoid nodules	Subcutaneous nodules over bony prominences, or extensor surfaces, or in juxta-articular regions, observed by a physician
6	Serum rheumatoid factor positive	
7	Radiographic changes	Radiographic changes typical of rheumatoid arthritis on hand and wrist radiographs, which must include evidence of erosions or periarticular osteopenia (osteoarthritis changes alone do not qualify)

A patient is said to have RA if he/she has satisfied at least four of these seven criteria. The first four criteria must have been present for **at least** 6 weeks.

Differential Diagnosis

SLE, Psoriatic arthritis, Infectious arthritis, Osteoarthritis, Sarcoidosis, Gout.

Treatment

1. Physical and occupational therapy—strengthen periarticular muscles, consider assistive devices.
2. Drugs
   - Aspirin or NSAIDs.
   - Intra-articular glucocorticoids.
   - Systemic glucocorticoids.
     - These drugs (NSAID and glucocorticoids) control of pain and inflammation, but drugs do **not** alter disease progression.
     - Long term use of NSAID can cause peptic ulcer.
   - Disease-modifying antirheumatic drugs (DMARDs):
     - These agents need to be started early in the course of disease (ideally within 3 months). The hallmark of these agents is their ability to halt progression of disease, such as the development of erosions. All of them take weeks to months to start working.
     - Methotrexate: **is first-line drug**, given as weekly low-dose oral or parenterally administered. Remission of rheumatoid arthritis on DMARD therapy has been described in approximately 20 per cent of those with early disease treated with methotrexate or sulphasalazine as single agents.
     - Sulfasalazine: is the other first line drug. *Side effects*: GI upset, depression, and reversible oligospermia.
     - Hydroxychloroquine: side effect is macular damage, and monitored by eye examination every 6 months to 1 year.
     - Gold therapy (either by IM or orally) takes 2-3 months to start acting. *Side effects* include rashes, thrombocytopenia, leukopenia, aplastic anemia and glomerulonephritis (nephrotic syndrome). About 60% of patients may be expected to benefit from gold therapy.
     - D-penicillamine can cause nephrotic syndrome.
   - Anti-cytokine therapy—TNF modulatory agents:
     - Etanercept: s/c administration, can cause demyelination
     - Infliximab: IV administration, risks include reactivation of TB
3. Surgery—may be considered for severe functional impairment due to deformity.

Prognosis

Poor prognostic features of RA:

1. Female sex
2. Insidious onset
3. Extra articular features e.g. **nodules**
4. Poor functional status at presentation
5. Rheumatoid factor positive
6. HLA DR4
7. X-ray: early erosions (e.g. after < 2 years)

Thyroid gland

- Introduce yourself & while shaking hands, note whether the palms are warm & sweaty.
- **Inspection** of the neck:

1. Swelling: multiple or single, diffuse or localized, site (medline or lateral), size, surface (smooth or nodular).
2. Ask the patient to swallow [tell the patient to sip a water if glass of water is present]
3. Skin: color, edema, scar, sinus, fistula, dilated veins, and pulsility.
4. Look for the JVP.
5. Scars of surgery (often missed by candidates).
6. Enlarged cervical lymph nodes.

- **Palpation** (always begin by palpating from behind):

1. Seat the patient comfortably, and the put the neck slightly extended position.
2. Comment first on exophthalmos.
3. While palpating the gland, ensure that there is a glass of water to swallow.
4. Palpate the skin for Temprature and Tenderness
5. Palpate the thyroid and note the following: - Size [measure it with tape measure] - specify the WHO grade. - Mobility. -Texture - simple or nodular (solitary or multiple)?
6. Palpate for extension behind the lateral border of sternocleidomastoid ms [by asking the patient to push his head against your hand the and feel for the otherside].
7. Palpate for extension downward by palpating the tracheal ring.
8. Palpate cervical lymph nodes.
9. Feel the carotid arteries.
10. Palpate for tracheal deviation.
11. Pemberton's sign (on raising the arms above the head, patients with retro-sternal goitres may develop signs of compressim, i.e. suffusion of the face)

- **Percuss** for retrosternal extension.
- **Auscultate** over the gland for bruit [by diaphragm over upper pole], carotid bruits.

Test sternomastoid function (this muscle may be infiltrated in thyroid malignancy).

- Thyroid function should then be assessed:

1. Eye signs:
   - Lid lag.
   - Exophthalmos.
   - Lid retraction (sclera visible above the cornea).
   - Extraocular movements.
2. Hands:
   - Pulse for tachycardia or atrial fibrillation.
   - Tremor.
   - Acropachy or clubbing.
   - Palmar erythema (thyrotoxicosis) & sweating.
   - Supinator = brachioradialis jerks (inverted in hypothyroidism).
   - Proximal weakness in the upper arm.
3. Skin: look for pretibial myxoedema.
4. Elicit the ankle jerks.

How would you grade the size of the goitre?

WHO grading of goitre:

Grade 0: No palpable or visible goitre.

Grade 1: Palpable goitre (larger than terminal phalanges of examiner's thumbs).

IA Goitre detectable only on pt Ipation.

IB Goitre palpable and visible with neck extended.

Grade 2: Goitre visible with neck in normal position.

Grade 3: Large goitre visible from a distance.

What is the significance of the thyroid bruit?

The thyroid bruit is almost pathognomonic of Graves' disease and occurs only rarely in patients with colloid goitres or other thyroid disorders.

Which is the best laboratory test to diagnose hyperthyroidism?

Serum TSH measurement is the single most reliable test to diagnose all common forms of hyperthyroidism, particularly in an outpatient setting.

What drugs are used in the treatment of thyrotoxicosis?

- Carbimazole.
- Methimazole.
- Propylthiouracil.

What are the disadvantages of antithyroid drugs?

- High rates of relapse once treatment is discontinued.
- Occasionally complicated by troublesome hypersensitivity reaction and very rarely by life-threatening agranulocytosis and hepatitis.

What is the single best clinical indicator for hypothyroidism?

Delayed ankle jerks.

What is the best laboratory indicator for hypothyroidism?

Elevated serum TSH levels. When there is a suspicion of pituitary or hypothalamic disease, the serum free T4 concentration should be measured in addition to serum TSH levels. Serum triiodothyronine concentrations are a poor indicator of hypo-thyroid state and should not be used.

What is the hazard in treating the elderly?

Rapid T4 replacement may precipitate angina and myocardial infarction. The starting dose in the elderly is 50/ag per day.

What are the cardiovascular manifestations of hypothyroidism?

1. Bradycardia.
2. Mild hypertension.
3. Pericarditis and pericardial effusion.
4. ↑ LDL, ↓ HDL levels.
5. Coronary artery disease.

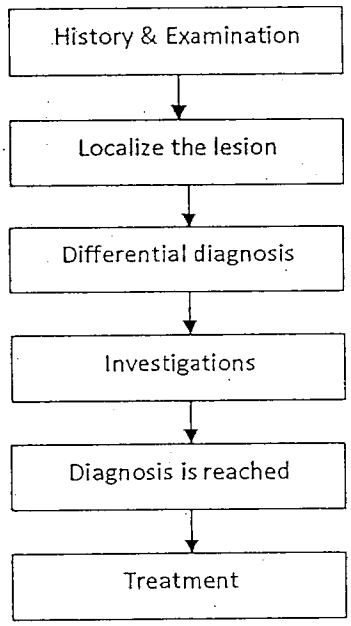
Thyroid Examination

Thyroid examination checklist					
Action	YES	NO	**Action**	YES	NO
Permission taken Stood on the Right side of bed			Temperature Tenderness		
Adequate Exposure			Palpate one lobe at time		
Position the patient			Give the pt glass of water		
Inspection			Ask the pt to swallow		
Stand at the end of the bed			Assess mobility		
Symmetrical or not			Test for extension behind SCM		
Swelling (midline or lateral)			Palpate the trachea		
Ask the patient to swallow			Palpate the LN		
JVP			**Percussion**		
Scar			Over the sternum		
Superficial veins			**Auscultation**		
Discoloration			Over the thyroid		
Palpation			**Ethics**		
Ask the patient to sit			Thank the patient		
Stand from behind			Thank the examiner		

An Introduction to Neurology

HOW NEUROLOGY IS WORKING?

1. The first step in the neurology is to LOCALIZE the lesion [By history, physical examination].
2. The second step is to formulate a DIFFERENTIAL DIAGNOSIS [based upon the diseases that affect the involved part of the nervous system and the patient's history and risk factors].
3. The 3rd step is to run INVESTIGATION TO REACH A DIAGNOSIS
   - Blood, CSF, Imaging, Biopsy
4. Diagnosis
5. Treatment Plan



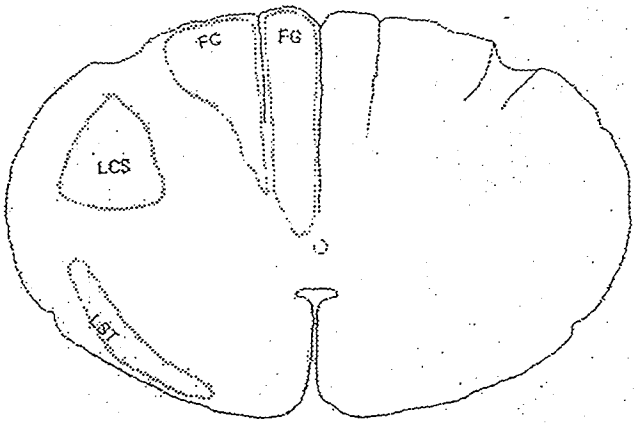
The Organization of the Nervous System

Central Nervous System (CNS)
- Brain
- Brain Stem--medulla, pons, midbrain
- Cerebellum
- Basal Ganglia--caudate, putamen, globus pallidus, subthalamic nucleus, substantia nigra
- Spinal Cord

Peripheral Nervous System (PNS)
- Nerves & nerve roots
- Ganglia

Spinal cord

ALL YOU NEED TO KNOW IS 3 TRACTS:
1. Dorsal Columns
2. Spinothalamic Tract
3. Corticospinal Tract

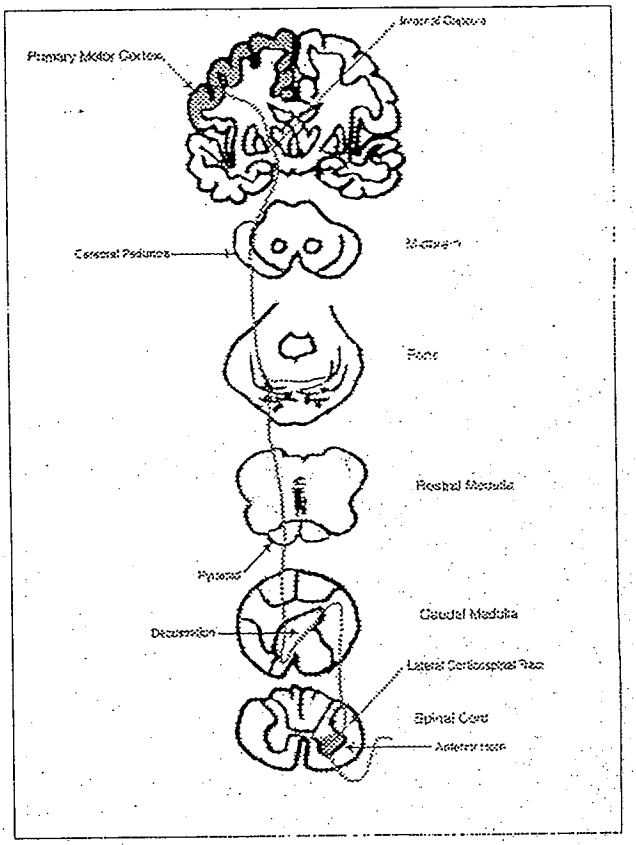


Lateral Corticospinal Tracts = Pyramidal System (Voluntary Movement)

Function: Voluntary movements

Lateral corticospinal tract = Upper motor neurons: motor neurons in the cerebral cortex and their axons descend through the subcortical white matter, internal capsule, brainstem, and spinal cord.

Motor unit includes: **the lower motor neurons** in the ventral horn of the spinal cord and their axons in the spinal roots and peripheral nerves, neuromuscular junction, and skeletal muscle.



In general:
- *Upper motor neuron dysfunction*: increased muscle tone (spasticity), brisk deep tendon reflexes, and Babinski sign.
- *Lower motor neuron dysfunction*: reduced muscle tone, diminished reflexes, and muscle atrophy.

Definitions
Tone is the resistance of a muscle to passive stretch which is measured at the joint
Spasticity: an increase in tone associated with disease of upper motor neurons. Spasticity is velocity-dependent, has a sudden release after reaching a maximum (the "clasp-knife" phenomenon), and predominantly affects the antigravity muscles (i.e., upper-limb flexors and lower-limb extensors).
Rigidity is increased tone associated with extrapyrmidal lesion that is present throughout the range of motion ("lead pipe") and affects flexors and extensors equally.

Motor Examination

Inspection	Expose the 2 limbs to be examined then Look for: 1. posture = deformity (e.g. pectus cavus) 2. Wasting 3. Fasciculation 4. Abnormal movements (convulsion, tremor, chorea, athetosis) 5. scars Try to provoke fasciculation
Palpation	For temperature and tenderness. Palpate for the ms bulks
Tone	1. Make sure that patient is pain free, relaxed, and not helping you. 2. You must look at the patient face while doing that.
Power	MRC scale
Reflexes	Deep: 1.Biceps 2. Triceps 3. Brachioradialis 4.knee 5. Ankle 6. clonus Reflexes are considered abnormal when are associated with other signs or they are asymmetrical Young people may have normal brisk reflexes Clonus can be un-sustained or sustained if more than 3 twitches.
	Superficial: 1. Babinski 2. Hoffman 3. Abdominal Absent Plantar occurs in 1.LMNL 2.Obese 3. Children. Babinski is normal in 1. Infants due to immature pyramidal system 2. Sleep.

The aim of motor examination is to decide whether the lesion is upper motor or lower motor neuron lesion

	UMNL	LMNL
Wasting	No	Wasted
fasciculation	No	Yes
Tone	Spastic	Decreased
Distribution of weakness	Pyramidal/regional	Distal/segmental
Clonus	+ve	-ve
Tendon reflexes	Hyperactive	Hypoactive/absent
Babinski's sign	Present	Absent

MRC (Medical Research Council) scale for muscle power

0 = no movement
1 = flicker or trace of contraction but no associated movement at a joint
2 = movement with gravity eliminated
3 = movement against gravity but not against resistance
4 = movement against resistance but not full power
5 = full power

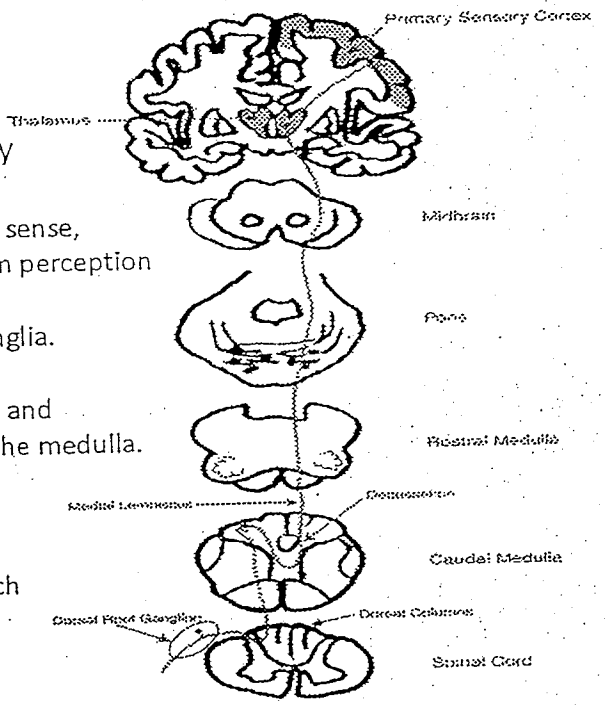
Reflexes

Upper limbs
Biceps (**C5**, C6)
Brachioradialis (C5, **C6**)
Triceps (**C7**, C8)

Lower limbs
Knee (**L3**, L4)
Ankle (**S1**, S2)

The reflex may be absent, diminished, normal, or exaggerated.

Sensory



• Dorsal Column-Medial Lemniscus Pathway (Position-Sense-Vibration)

Function: it mediates vibration sensation, position sense, fine touch, 2 points tactile discrimination and form perception

First order neuron is located in the dorsal root ganglia.

Second order neurons are located in the **medulla**, and here the fibers **decussate** to the opposite side of the medulla.

Third order neurons are found in the thalamus, they project through the posterior limb of the internal capsule to the postcentral gyrus which is the primary somatosensory cortex

Note: In sensory pathways, usually the second order neuron sends its axon across the midline.

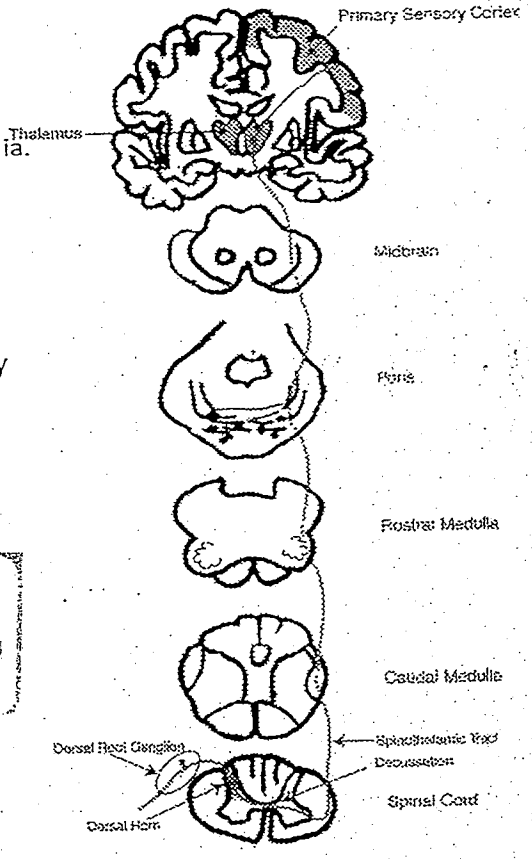
• Spinothalamic Tracts (Pain-Temperature)

Function: mediates pain and temperature sensation

First order neuron is located in the dorsal root ganglia.

Second order neurons are located in the **dorsal horn**, and here the fibers **decussate** to the opposite side of the cord.

Third order neurons are found in the thalamus, they project through the posterior limb of the internal capsule to the postcentral gyrus which is the primary somatosensory cortex



Coordination (Cerebellum)

Coordination between agonist and antagonist muscle done by the basal ganglia during rest (static tremors) and by the cerebellum during active movement (kinetic tremor)

Signs of cerebellar diseases:

1. Nystagmus.
2. Speech is scanning.
3. Loss of coordination. In the upper limbs is tested by the finger to nose tests and alternating hand movements. In the lower limbs is tested by heel-toe test, gait, and Romberg's test.
4. Hypotonia.
5. Absent reflexes or pendular reflexes.
6. Lack of co-ordination of gait = ataxia (wide based gait)- patient tends to fall towards the side of the lesion.

Causes of UMNL:

1- CVA
2- Multiple sclerosis
3- Space occupying lesion
4- Vasculitis
5- Trauma→ Subdural hematoma
6- Brain abscess

Causes of LMNL:

1- Trauma
2- ALS
3- Poliomyelitis
4- Spinal artery thrombosis

The finding of an extensor plantar response with an absent ankle reflex suggests a mixed UMN and LMN lesion. There are 3 classical causes.

1. In vitamin B12 deficiency, both peripheral neuropathy and subacute combined degeneration of the cord may occur.
2. Friedreich's Ataxia.
3. Diabetic amyotrophy
4. Others: Syringomelia, Amyotrophic lateral sclerosis, cervical myelopathy, and tabes dorsalis.

Causes of spastic paraparesis or paraplagia

1- Trauma
2- Multiple sclerosis
3- Friedreich's ataxia
4- HIV
5- Spinal cord tumor

Upper Limbs Examination checklist

Action	YES	NO
Permission taken		
Stood on the Right side of bed		
Adequate Exposure		
Inspection		
Posture		
Wasting		
Scars		
Abnormal movements		
Fasciculation		
Palpation		
Tenderness		
Consistency		
Motor		
Tone (shoulder, elbow, wrist)		
Power		
Shoulder Abduction		
Shoulder Adduction		
Elbow flexion		
Elbow extension		
Wrist flexion		
Wrist extension		
Short & Long flexor (grip)		
Fingers abduction & adduction		
Comment using MRC Grade		

Action		YES	NO
Reflexes			
Deep	Biceps		
	Triceps		
	Brachioradialis		
If absent test with reinforcement			
Superficial (Hoffman's test)			
Sensory			
Pain			
Temperature			
Light touch			
Joint position			
Coordination			
Finger to nose test			
Dysdiadochokinesia			
Ethics			
Cover the patient			
Thank the patient			
Thank the examiner			

Lower Limbs Examination checklist

Action	YES	NO
Permission taken		
Stood on the Right side of bed		
Adequate Exposure		
Inspection		
Posture		
Wasting		
Scars		
Abnormal movements		
Fasciculation		
Palpation		
Tenderness		
Consistency		
Motor		
Tone (hip, knee, ankle)		
If hypertonia test for Clonus in the patella and ankle		
Power		
Hip flexion		
Hip extension		
Hip abduction		
Hip adduction		
Knee flexion		
Knee extension		
Ankle dorsiflexion		
Ankle plantar flexion		
Eversion and Inversion		
Comment using MRC Grade		

Action		YES	NO
Reflexes			
Deep	Knee		
	Ankle		
If absent test with reinforcement			
Superficial (Plantar reflex)			
Sensory			
Pain			
Temperature			
Light touch			
Joint position			
Coordination			
Heel to shin test			
Gait			
Walking			
Heel-Toe			
Romberg's test			
Ethics			
Cover the patient			
Thank the patient			
Thank the examiner			

Cranial Nerves

The nuclei of the cranial nerves:
CN I and CN II: nuclei are located in the brain.
CN III and CN IV: nuclei are located in the midbrain.
CN V, CN VI, CN VII, and CN VIII: nuclei are located in the pons.
CN XI, CN X, CN XI, and CN XII: nuclei are located in the medulla.

Olfactory nerve, the first cranial nerve (**CN1**), it mediates olfaction (smell).
Pathway: the receptors are located in the nasal cavity, and fibers from these receptors pass through the cribriform plate, and form the olfactory bulb, and from there to the temporal lobe.

Optic nerve (CN II)

Oculomotor nerve (CNIII)
Pathway: the nucleus is located in the midbrain, passes through the cavernous sinus, and enters the orbit through the superior orbital fissure
General characteristics: the oculomotor nerve moves the eye, constricts the pupil, accommodates and converges.

- The oculomotor nerve supplies all of the muscles of the eye except superior oblique muscle and lateral rectus muscle.
  - **Medial rectus muscle** adducts the eye.
  - **Superior rectus muscle** elevates and adducts the eye.
  - **Inferior rectus muscle** depresses, and adducts the eye.
  - **Inferior oblique muscle** elevates, and abducts the eye.
  - **Levator palpebrae muscle** elevates the upper eyelids.
- **Edinger-Westphal nucleus** projects preganglionic parasympathetic fibers to the **ciliary ganglion** of the orbit through the CN III, and from the ciliary ganglion postganglionic parasympathetic fibers projects to the sphincter muscle of the iris (**miosis**) and the ciliary muscles (**accommodations**).

Oculomotor palsy
Causes: DM, Infarction, Trauma, Tumor, Aneurysm, raised ICP.
Clinical presentation:

- diplopia • complete ptosis • outwardpupil dilatation (Mydriasis)
- paralytic divergent squint with eyes looking downward & Note: Compressive lesions of oculomotor nerve usually dilate the pupil; ischemic lesions (e.g., in diabetes), which involve the central portion of the nerve, usually do not.

Trochlear nerve (CN IV)
It is a pure motor nerve that supplies superior oblique muscle, which function is to depress and abduct the eye.**Pathway:** the nucleus is located in the midbrain; it passes through the cavernous sinus, and enters the orbit through the superior orbital fissure.
Superior oblique (fourth, or trochlear nerve) palsy: (most common cause is trauma)
Vertical diplopia which is most marked on looking downward and inward and corrected by **tilting the head** away from the side of the lesion.

Trigeminal nerve (CN V)

Anatomy: the nucleus of the fifth nerve is located in the pons.

Sensory division: it supplies sensation to the face, mucous membranes of the nasal and oral cavities, and deep structures of the head.

- Ophthalmic: leaves the skull through superior orbital fissure.
- Maxillary: leaves the skull through foramen rotundum.
- Mandibular: leaves the skull through foramen ovale.

Motor division supplies muscles of mastication (i.e. temporalis, masseter, lateral, and medial pterygoids) through the Mandibular nerve

Lesions of the fifth cranial nerve:

- **Loss of sensation** from the face and mucous membranes, from the same side.
- **Loss of corneal reflex** [afferent→ ophthalmic branch of CN V, & efferent → CN VII]
- Flaccid paralysis of the muscles of mastication.
- Deviation of the jaw to the weak side

Abducent nerve (CN IV)

It is a pure motor nerve which its nucleus is located in the pons. The nerve supplies lateral rectus muscle, which abducts the eye.

Abducens (sixth nerve) palsy: (most common cause is raised intracranial pressure)

- Medial deviation of the eye.
- Horizontal diplopia

Glossopharyngeal nerve (CN IX)

Motor to → stylopharyngeus & superior constrictor.

Sensory to → pharynx - Tonsils - postior 1/3 of tongue

Taste from posterior 1/3 of the tongue

Lesion: ipsilateral loss of taste, ipsilateral loss of pharyngeal reflex

Vagus nerve (X)

Motor to soft palate, pharynx, & larynx

Sensory from Skin over external auditory meatus and mucous membrane of GIT and the respiratory tree.

Accessory nerve (XI)

It's formed of 2 parts:

Cranial part → Runs with the vagus & shares the innervation of the soft palate & pharynx

Spinal part → Sternomastoid & trapezius

Hypoglossal nerve (XII)

It is the motor supply for muscles of the tongue

In unilateral lesion it will point to the side of the lesion

Note: Cranial nerves XI, X, XI, exit the skull from the jugular foramen, while the hypoglossal nerve exits from the hypoglossal foramen.

Facial nerve (CN VII) MOST IMPORTANT CRANIAL NERVE

Course: The nucleus of the facial verve lies in the pons, the efferent fibers loop around 6th cranial nerve nucleus and leave the pons from its lateral aspect to enter the Cerebellopontine angle, and then enter the geniculate ganglion [from which superficial petrosal nerve → pterygopalatine ganglia → Parasympathetic for lacrimal glands] the facial nerve enters internal auditory meatus where it runs in the **facial canal** which lies in the posterior wall of tympanic cavity = middle ear where it gives [1. nerve to stapedius which supplies stapedius ms which is important for acoustic reflex, 2. chorda tympani which runs foreword in the medial aspect of the upper part of the tympanic cavity and leaves trough petrotympanic fissure → joins the lingual nerve → submandibular ganglion → submandibular and sublingual salivary glands and also taste sensation from anterior 2/3 of the tongue] the facial nerve then emerges from the **stylomastoid foramen** and passes anteriorly trough the substance of parotid gland dividing it into superficial and deep parts and then gives of :

1. Temporal
2. Zygomatic
3. Buccal
4. Mandibular
5. Cervical

Summery of the course:
The nucleus lies in the pons→ the nerve leaves from the lateral surface of the pons→ Cerebellopontine angle → relay in the geniculate ganglia [which sends sup. Petrosal N] → enters the internal auditory meatus→ trough the facial canal [in canal gives Nerve to stapedius and Chorda tympani] leaves the skull by emerging from stylomastoid foramen → enters parotid gland and branches to supply the face

Localization of the abnormality is based on the neuroanatomy

Clinical Features	Site
6th and 7th cranial nerves palsies	Pons
5th, 7th, 8th + cerebellar signs	Cerebellopontine angle tumor
Lacrimation involved	Lesion before int. audit. meatus
Hyperacusis or loss of Taste	Lesion in the facial canal
No Hyperacusis or loss of Taste	Lesion at the stylomastoid foramen or in the parotid gland

What are the branches of the facial nerve?

1. Greater superficial petrosal nerve (supplies lacrimal glands).
2. Nerve to stapedius muscle.
3. Chorda tympani (supplies taste to anterior two thirds of tongue, submaxillary and sublingual glands).
4. Motor branches (5) (exit from the stylomastoid foramen).

Tests

Test	Muscle	Nerve supply
Raising of eye brows	Frontalis	Temporal br
Drawing eyelids downward (frown)	Curragator	Temporal br
Closure of the eye	Orbicularis oculi	Temporal and zygomatic
Smile	zygomaticus major	zygomatic nerve
Elevation of the lip angle	Levator anguli oris	buccal
Closure of mouth and lip purse and Whistle	Orbicularis oris	buccal and Mandibular br

Examination

Action	YES	NO
Permission taken		
Stood on the Right side of bed		
Inspection		
Comment of the symmetry		
Wrinkles over the forehead		
Lid retraction		
Flattening of the naso-labial fold		
Depression of the mouth angle		
Deviation of the angle of the mouth		
Tests		
Elevate your eye brows		
Depress your eye brows		
Close your eyes and don't let me open them [comment on Bell's phenomena]		
Show me your teeth		
Purse your lips and blow your cheeks		

Action	YES	NO
Tips		
Look for external auditory meatus		
Palpate the parotid gland		
Say that you would like to		
Test for Corneal reflex		
Test for Taste in the Ant 2/3 of tongue [chorda tympani involvement]		
Examine internal ear by otoscope		
Would like to ask the pt about intolerance to high pitched sounds Hyperacusis [Paralysis of stapedius ms]		
Ethics		
Thank the patient		
Thank the examiner		

How to reach diagnosis?
History of URTI is important since the most common cause is Bell's palsy which is known to occur after URTI, also history of pain preceding or accompanying the weakness is suggestive
Ix→ RBS and FBS to exclude diabetes
→ Radiological Ix: CT and MRI

Differences between UMNL and LMNL in facial nerve palsy:
Forehead is spared in UMNL

Is the facial nerve motor or sensory?
It's a motor nerve that supplies the muscles of facial expression

Causes of unilateral lower facial nerve palsy

1. Bell's palsy is the most
2. Diabetes
3. Ramsay-Hunt syndrome: is caused by herpes zoster infection of geniculate ganglion; distinguished from Bell's palsy by a vesicular eruption in pharynx and external auditory canal, and by frequent involvement of eighth cranial nerve
4. Pontine hemorrhage
5. Cerebellopontine angle tumor
6. OM
7. Parotid tumor

Causes of bilateral lower facial nerve palsy

1. Fisherman syndrome (part of GBS)
2. Heerfordt's syndrome (part of Sarcoidosis)
3. Diabetes
4. Leprosy
5. Lyme disease

Causes of upper facial nerve palsy

1. Stroke
2. Tumor
3. MS
4. Brain Abscess

BELL'S PALSY

Most common cause of facial paralysis, affecting 1 in 60 persons over a lifetime.

Epidemiology: male = female, occurs in all ages, and all times of the years.

Etiology:

The lesion is within the facial canal; the cause is unknown but may be due to reactivation of latent herpes simplex virus 1 infection.

Clinical presentation:

- Weakness evolves over 12–48 h, sometimes preceded by retroaural pain.
- Hyperacusis may be present.
- No sensory loss (except for tatse)
- Full recovery within several weeks or months in 80%; incomplete paralysis in first week is a favorable prognostic sign.

Diagnosis can be made clinically in pts with (1) a typical presentation, (2) no risk factors or preexisting symptoms for other causes of facial paralysis, (3) no lesions of herpes zoster in the external ear canal, and (4) a normal neurologic examination with the exception of the facial nerve

Management

1. Physiotherapy: massage, electrical stimulation, splint to prevent drooping of the lower part of the face.
2. Protection of the eye with lubricating eye drops and a patch during sleep.
3. A short course of Prednisolone 60 to 80 mg daily for frist 5 days and tapered over the next 5 days (should be given within 48 hours of onset).
4. Aciclovir 400 mg 5times/day for 10 days(should be started within 3 days)
5. Vitamin B12 injections of 500 to 1,000 mcg given every one to two days

Cranial nerves examination

• *I: olfactory*	Pt closes eyes, test one nostril at time, use 3 common smells, avoid irritating smell(ammonia)
• *II: optic*	Test vision with Snellen chart or Counting method. Perform ophthalmoscopic exam of fundi. Test visual fields by confrontation test.
• *III, IV, VI: oculomotor, trochlear, abducens*	Inspect eyelids for drooping. Inspect pupils' size for equality & direct & consensual response to light & accomodation. Test extraocular movements.
• *V: trigeminal*	Inspect face for muscle atrophy and tremors. Palpate jaw muscles for tone and strength when pt clenches teeth Test superficial pain and touch sensation in each branch Test corneal reflex
• *VII: facial*	Inspect symmetry of facial features w/various expressions (smile, frown, puffed cheek, wrinkled forehead, purse lips and blow out, show teeth, squeeze eyes shut) Look at the external ear and examine the parotids. Test for salt, and sweat taste.
• *VIII: vestibulocochlear*	Test sense of hearing with whisper tests Compare bone and air conduction of sound Test for lateralization of sound
• *IX: glossopharyngeal*	Test ability to identify sour and bitter tastes Test gag reflex and ability to swallow
• *X: vagus*	Inspect palate and uvula for symmetry with speech sounds and Observe for swallowing difficulty
• *XI: spinal accessory*	Test trapezius muscle strength (shrug shoulders) Test SCM strength (turn head to each side)
• *XII: hypoglossal*	Inspect tongue in mouth and while protruded for tremors, and atrophy

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